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THERAPEUTIC CONSIDERATIONS IN THE MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION

TECHNIC OF ENTEROSTOMY AND A FURTHER ACCOUNT OF DECOMPRESSION BY THE EMPLOYMENT OF SUCTION SIPHONAGE BY NASAL CATHETER

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PHYSIOLOGICOPATHOLOGIC CONSIDERATIONS

The work of Hartwell and Hoguet [1] establishing the efficacy of subcutaneous administration of saline solution in definitely prolonging the lives of dogs with high intestinal obstruction lent considerable impetus to the experimental investigation of obstruction of the bowel. Unfortunately, a like interest in the field has not been generally manifest among clinicians, with the unhappy effect that results in the management of acute intestinal obstruction have not kept astride with increases in knowledge concerning the subject.

The experimental work of the decade following the observation of Hartwell and his associates dealt largely with the nature of the toxin in high intestinal obstruction and a study of the chemical alterations in the blood attending obstruction of the bowel. The numerous experimental studies of Haden and Orr contributed materially to a better understanding of the chemical changes attending obstruction of the upper part of the intestine. Investigations concerning the causes of death in obstruction of the bowel have not proved especially fruitful. A number of facts have been learned, but many things remain to be explained.

It is now known that the elevation of blood urea, decrease in plasma chlorides and increased combining power of the blood for carbon dioxide obtain with regularity only in high intestinal obstructions, but not sufficiently early enough to be of diagnostic value. Saline solution acts like a specific in high obstructions only, and not as an antidote or detoxifying agent, but as a substitute for important fluids lost by vomiting. The causes of death in low and midintestinal obstructions are not so well

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1. Hartwell, J. A., and Hoguet, J. P.: Experimental Intestinal Obstruction in Dogs with Special Reference to Cause of Death and Treatment with Large Amounts of Normal Saline Solution, J. A. M. A. **59**:82 (July 13) 1912.

understood but appear in some degree to be correlated with the function of the different levels of the bowel.[2]

No concrete proof exists of the absorption of a toxin in intestinal obstruction foreign to the normal bowel. On the contrary, there are many bits of evidence that indicate that such abnormal absorption in the presence of an obstructed but viable bowel does not occur. It is to be freely conceded, however, that failure to demonstrate objectively such abnormal absorption does not constitute a complete denial of its occurrence. At the same time, however, it indicates that this is not the central tenet of the problem.

Surgeons have long known that a well performed enterostomy will save the majority of persons with late simple obstruction, but that an attack directly on the obstruction is extremely hazardous to the patient's life. Enterostomy is life-saving in such instances, not because it drains off a potent toxin that threatens the organism but because it relieves tension within the bowel, restores the normal blood supply, allows the continuance of absorption from the bowel (which practically ceases in obstruction) and permits the automatic establishment of the continuity of the bowel in the absence of a persistent intrinsic obstruction below.

Instances of strangulation obstruction in which the blood supply to the bowel is compromised present additional features beside the block in the bowel, viz., loss of blood incident to greater occlusion of the veins than of the arteries to the segment concerned and a bowel which will become nonviable with persistence of the strangulating mechanism. If the veins to a segment of bowel from 3 to 5 feet in length are tied off in the dog, the animal will die of hemorrhage (into the bowel) in from four to five hours. If an encirclement ligature is placed around the bowel and its mesentery, simulating more closely clinical types of strangulation obstruction, the animal will survive for about fifteen hours, and the loss of blood is shown to be an important item in its death. If the arteries alone are tied, the animal survives for about twenty hours. The loss of blood or blood plasma is minimal, the necrosis of the wall of the bowel being the signifiant item in terminating the dog's life (Scott and Wangensteen[3]).

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2. Wangensteen, O. H., and Leven: Correlation of Function with Cause of Death Following Experimental Intestinal Obstruction at Varying Levels, Arch. Surg. **22**:658 (April) 1931, fig. 4.

3. Scott, H. G., and Wangensteen, O. H.: Studies in Strangulation Intestinal Obstruction: 1. Length of Life Following Various Types of Strangulation Obstruction in Dogs, Proc. Soc. Exper. Biol. & Med. **29**:424, 1932; 2. Blood Pressure Changes Correlated with Time, Length and Type of Intestinal Strangulation in Dogs, ibid. **29**:428, 1932; 3. Effect of Intravenous Injections of Peritoneal Fluids Recovered from Dogs Dying of Experimental Intestinal Strangulations, ibid. **29**:559, 1932; 4. Blood Losses in Experimental Intestinal Strangulations and Their Relationship to Degree of Shock and Death, ibid. **29**:748, 1932.

ESSENTIALS OF DIAGNOSIS

The importance of early recognition of abdominal disorders of an acute nature necessitating surgical intervention is generally recognized. A high degree of correlation exists between the ultimate mortality figure and the time intervening between the onset of the disaster and the institution of adequate treatment. Acute intestinal obstruction is no exception to this rule, and the persistently high mortality attending its surgical relief reflects only too plainly the effects of late diagnosis.

In hospitals where the resident and attending staff alike are "obstruction minded," the supervention of acute intestinal obstruction during convalescence from operation is immediately recognized. The intern knows that the occurrence of crampy intermittent abdominal pain, which the patient is likely to term "gas pains," is identified as intestinal colic by the concomitant presence of loud intestinal borborygmi heard with the stethoscope at the acme of the pain. He knows that none of the other colics to which the abdomen is heir is characterized by the simultaneous occurrence of loud intestinal noises at the height of the cramp. After the establishment of the presence of intestinal colic, it only remains to determine whether the pain is due to mechanical obstruction, acute enterocolitis, a type of abdominal allergy or food poisoning, or to the presence of simple "belly-ache." On the basis of other evidence, such as the presence or absence of nausea and vomiting, diarrhea and intestinal distention, a reliable deduction can ordinarily be made.

An agent of great value in this interpretation is a single plate made of the abdomen with the patient supine. It reveals the presence of visible gas in the small intestine, a sign significant of intestinal stasis in the adult; it determines also the degree of distention of the bowel.

There are four causes commonly seen to contribute to delay in the diagnosis of acute intestinal obstruction:

1. Failure to appreciate that simple obstruction of the bowel is not accompanied by tenderness or rigidity of the abdominal wall.
2. Belief that the expulsion of gas or feces with an enema militates against obstruction of the bowel.
3. Assuaging of pain with morphine.
4. The deception of apparently effectual catharsis in partial obstructions.

Patients with strangulation types of obstruction almost invariably present local tenderness and rigidity of the abdominal wall due to the escape of hemorrhagic fluid into the peritoneal cavity. Their complaint and physical findings are otherwise those of intestinal colic as in simple obstruction. Patients with strangulation obstruction exhibit early slight quickening of the pulse incident to loss of blood into the infarcted segment, and early rises of fever to 100 and 101 F. are usual. There is no disturbance of the general condition of the patient early in simple obstruction. Chemical alterations in the blood occur with regularity only

in high obstructions and are usually not observed until forty-eight hours or more has elapsed following the onset of the obstruction. Leukocytosis may occur in either simple or strangulation obstruction incident to the dehydration of vomiting.

A patient complaining of intermittent crampy pain attended by nausea and vomiting without manifesting local tenderness or rigidity of the abdomen should be suspected of having simple intestinal obstruction. Should pain continue despite the effectual return of gas and feces with enemas in the presence of visible distention of the small intestine, on roentgen examination the diagnosis of intestinal stasis can reasonably be made. The occurrence of loud intestinal borborygmi significant of

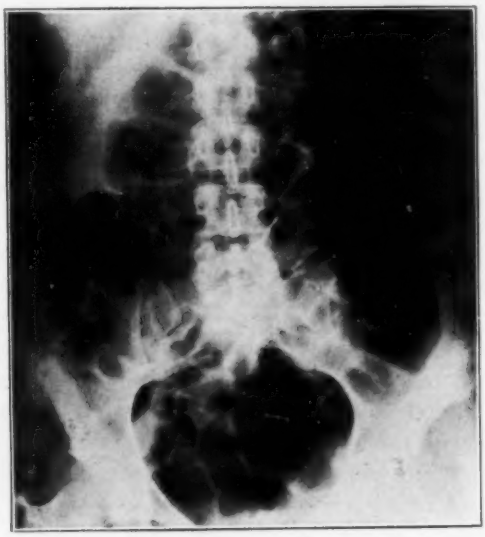

Fig. 1.—Roentgenogram of the abdomen of a patient with "functional spastic ileus." The distention is limited to the colon. This patient had been operated on six times previously for acute intestinal obstruction.

increased peristaltic activity of the bowel at the height of the pain establishes the stasis as being mechanical in nature. The stethoscope is an important agent in the diagnosis.

Elsewhere [4] I have described a group of cases in which no organic obstruction is present which simulate the clinical picture of acute mechanical obstruction very closely. The gaseous distention, however, is confined entirely to the colon and the stomach. The history and the gaseous distention as visualized on the x-ray film (fig. 1) serve to identify the

4. Wangensteen, O. H.: Elaboration of Criteria upon Which the Early Diagnosis of Acute Intestinal Obstruction May Be Made, with Special Consideration of the Value of X-Ray Evidence, Radiology **17**:44 (July) 1931.

Fig. 2.—Roentgenogram showing distention of the colon due to carcinoma of the sigmoid flexure. There was no distention of the small intestine, owing to the competence of the ileocecal sphincter. At operation the cecum was found to be gangrenous due to the tension.

Fig. 3.—Roentgenogram showing incomplete adhesive obstruction of the small intestine. Gas is present in the colon, but the latter is not distended. Decompression was effected by suction siphonage through a nasal catheter.

condition. The roentgen features of this condition resemble rather closely the colonic distention of carcinoma of the sigmoid flexure with obstruction (fig. 2). Partial adhesive obstructions in which gas is already present in the colon (fig. 3) are not to be mistaken for this variety of functional spastic ileus. The gaseous distention of the small intestine differentiates the two.

REMEDIAL AGENTS

Early release of the obstruction is the keynote of successful therapy of acute intestinal obstruction. In the majority of instances, immediate recourse is to be had to surgical measures of relief. Strangulation varieties of intestinal obstruction are always to be dealt with surgically as quickly as possible because of the threatened devitalization of the obstructed bowel. Some types of simple obstruction, especially those in which decompression of the bowel (enterostomy) serves to reestablish its continuity, can be satisfactorily treated by nonoperative means (suction siphonage by nasal catheter).

Saline Solution.—This solution exhibits the virtues of a specific only in high obstructions. Clinically, the only types which are indisputably known to be "high" are those at the pyloric outlet of the stomach and those which occasionally obtain temporarily at the stoma after the performance of gastro-enterostomy or gastric resection with anastomosis (Polya). In such instances, the surgeon feels safe in observing expectant treatment, replacing the fluid lost from the stomach by the generous administration of saline solution. In all obstructions, adequate administration of fluid is important, but the widespread impression that saline solution rehabilitates patients with acute intestinal obstruction is to be corrected. Though salt and water are important agents in combating the dehydration incident to persistent vomiting from obstruction, they do not accomplish miracles. Release of tension by decompression of the bowel in simple obstruction and early release of the constricting mechanism in strangulation obstruction are the ends to be attained.

During the hour that preparations are being made in the operating room, physiologic solution of sodium chloride should be freely administered (from 2 to 3 liters) by the intravenous and subcutaneous routes. In those instances in which the roentgenographic film indicates that the degree of intestinal distention is not great, more time may be consumed to replace adequately the fluid lost by vomiting.

Blood Transfusion.—For the poor surgical risk, transfusion is always a great boon. In simple obstruction, the transfusion of blood, just as the generous use of saline solution, fortifies the patient in some measure for the ordeal that he must undergo. Only in strangulation

types of obstruction, in which the loss of blood is frequently a significant item, is transfusion urgently indicated. In any patient with this type of obstruction presenting a high pulse rate and low blood pressure, transfusion is to be performed before operation. The adequate replacement of the blood lost is also important. The shock features of strangulating varieties of obstruction are synonymous with loss of blood, and shock in the healthy adult at any rate is not precipitated by lesser losses of blood than from 800 to 1,000 cc. In the ill patient, lesser losses are probably more telling. Nevertheless, transfusions for blood lost must be adequate.

Recently an infant of 18 months was observed at the University Hospital with an intussusception of about fifty hours' duration. The pulse was weak and extremely rapid, the rate being about 200. There was no manifest improvement after the subcutaneous and intravenous infusion of 700 cc. of saline solution. After the transfusion of 250 cc. of blood, the patient's condition improved remarkably. The pulse fell to 160, and reduction of a compound intussusception (entero-enteric and ileo colic) together with excision of a Meckel's diverticulum at the apex of the intussusception was accomplished without event, the patient making a satisfactory convalescence.

Suction Siphonage by Nasal Catheter.[5]—I have long employed conservative means in dealing with instances of functional spastic ileus, previously described, but was first led to employ drainage by suction in the treatment of acute mechanical obstruction in the following case:

E. J., a woman, aged 72, was admitted to the hospital with an obstruction of seventy-two hours' duration, and operation obviously was danger. The systolic blood pressure was 80 mm. of mercury, and the patient was badly dehydrated. Past experience with cases of this sort had taught that saline solution alone, even when administered for some considerable period of time, did not improve the patient materially as a surgical risk. The details of the apparatus and the manner of its employment are shown in the accompanying sketch (fig. 4).

The roentgen film (fig. 5 *A*) showed that considerable distention of the entire small intestine was present. It was felt that if a lessening of the distention could be accomplished the risk of the operation could be definitely decreased. After the insertion of the nasal catheter and the commencement of suction, the crampy pain of which the patient complained ceased at once. After eight hours' suction (fig. 5 *B*), during which time 200 cc. of fluid and 800 cc. of gas were aspirated, the distention of the small intestine was considerably reduced and the patient's condition was equally improved. After forty hours' suction, there was complete disappearance of gaseous distention (fig. 5 *C*). Because of the conjectured persistent nature of the obstruction (thought to be carcinoma of the cecum), operation was done. A stricture of the terminal ileum, probably syphilitic in nature, was

5. Wangensteen, O. H.: The Early Diagnosis of Acute Intestinal Obstruction with Comments on Pathology and Treatment with a Report of a Successful Decompression of Three Cases of Mechanical Bowel Obstruction by Nasal Catheter Suction Siphonage, West. J. Surg. **40**:1 (Jan.) 1932; Tr. West. S. A., 1931.

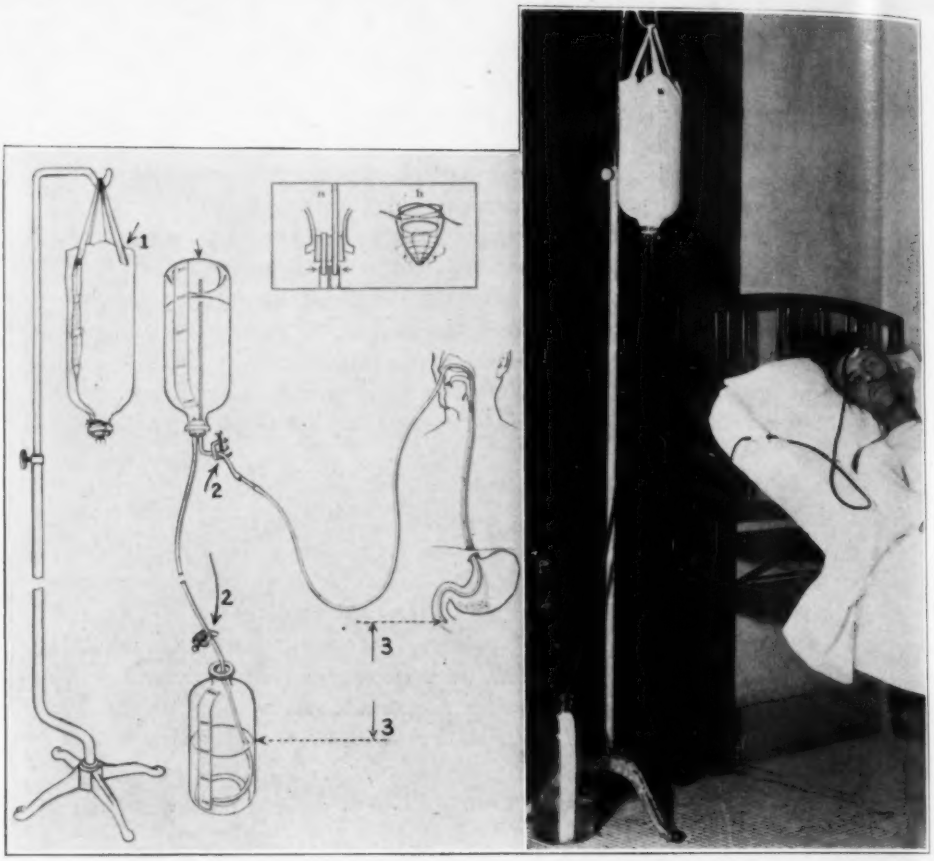

Fig. 4.—Sketch of the apparatus employed for suction siphonage through a nasal catheter, and a photograph of the apparatus in use. In the sketch, *1* indicates the canvas holder for the upper bottle; *2*, the clamp, to be used when the nasal tube is removed or the lower bottle emptied; *3*, the vertical distance—the degree of negative pressure in the nasal tube varying directly with this distance (2 feet, 6 inches has been found satisfactory—(*a*) to the lower bottle and (*b*) to the patient.

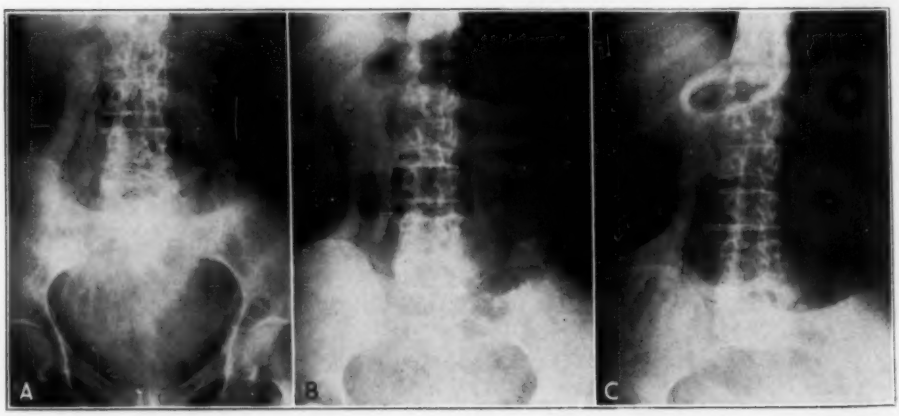

Fig. 5.—Roentgenograms of the abdomen of Mrs. E. J., 72 years old. *A* was taken on admission to the hospital with seventy-two hours' acute obstruction. The entire small intestine is distended. *B* was taken after eight hours' suction with a nasal catheter, during which time 200 cc. of fluid and 800 cc. of gas were aspirated. *C* was taken after forty hours' suction, showing complete decompression.

found.[6] The stricture was divided in the longitudinal axis of the bowel, the strictured area was invaginated into the cecum and a proximal enterostomy was done, following which the patient convalesced uneventfully.

Since then, successful decompression without operation has been done on seven patients with acute adhesive obstruction.

L. F., a boy, aged 9 years, was admitted to the hospital on Oct. 13, 1931. He had been operated on three years previously at the University Hospital for acute suppurative appendicitis with peritonitis. On his admission with acute obstruction, he gave a history of vomiting and intermittent colicky pain of forty-eight hours' duration. There was no rebound tenderness or abdominal rigidity. The roentgen film (fig. 6*A*) showed considerable distention of the upper part of the intestine. Immediately after the institution of suction the pain stopped, and a film taken three hours later (fig. 6*B*), at which time 550 cc. of fluid and 650 cc. of gas had been aspirated, showed complete disappearance of the gaseous distention of the small intestine. After twenty hours' suction (fig. 6*C*), no gaseous shadows were visible in the abdomen. Since the institution of suction, 1,625 cc. of fluid and 2,250 cc.

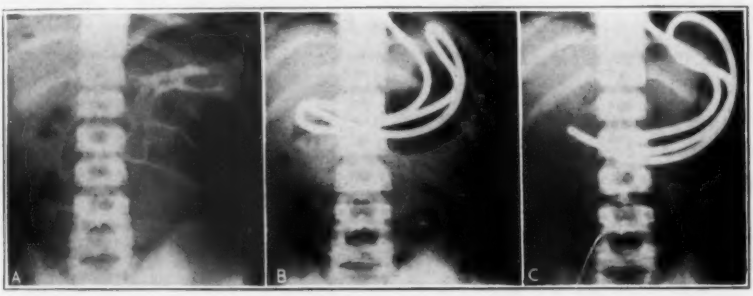

Fig. 6.—Roentgenograms of the abdomen of L. F., 9 years old. *A* was taken on admission to the hospital with a forty-eight hours' acute obstruction. *B* was taken after three hours' suction, when 550 cc. of fluid and 650 cc. of gas had been aspirated. *C* was taken twenty hours after commencement of suction; 1,625 cc. of fluid and 2,250 cc. of gas were aspirated meanwhile.

of gas had been recovered by aspiration. The suction was then discontinued, and the patient was permitted water by mouth, followed by clear liquids, and gradually solid food was allowed. He was permitted to be up and about, but it was felt that since he had previously had lesser attacks of the same sort the obstructive mechanism should be sought out and freed. Fifteen days after his admission, laparotomy was performed and an obstructive band in the midportion of the small intestine was found acutely angulating the bowel and binding it down to the root of the mesentery. The band was divided and the bowel released; the patient was dismissed from the hospital fourteen days later.

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6. Strictures of the lower portion of the small bowel behave peculiarly occasionally. I have recently observed an instance of intermittent obstruction of a subacute nature due to a stricture of the terminal ileum that would not admit the little finger. Still the patient was well without obstructive features for long intervals.

Successful decompression with suction siphonage by a nasal catheter without subsequent operation on the obstructive mechanism has been done in five patients with acute adhesive obstruction.

Mr. C., aged 26, presented acute adhesive obstruction seven days after an appendectomy for acute suppurative appendicitis. Enemas proved ineffectual in relieving his pain. Immediately after starting the suction, he had relief from pain. After seventy-two hours' suction, the bowel was completely decompressed and water was permitted by mouth.[7] The patient was subsequently discharged well.

Mr. B., aged 41, was admitted to the hospital with acute intestinal obstruction after having been successfully operated on elsewhere for the relief of acute obstruction about a month previously. The obstruction was only partial as shown by the persistent presence of gas in the colon. Suction siphonage by nasal catheter alone sufficed to decompress the bowel, and the patient was dismissed with the instruction that should he continue to have attacks of pain and vomiting it would be wise to establish the uninterrupted continuity of the bowel by severing the adhesive constricting bands at operation.

After the performance of enterostomy for the relief of adhesive obstruction, it is rarely necessary to have to deal later with the obstructive mechanism. Despite its persistent presence, just as mysteriously as the obstruction was initiated, so after effectual decompression the intestinal current goes on uninterrupted, and the patient suffers no ill effects from the continued presence of the adhesive bands. In the presence of universal adhesions causing continuous pain or intermittent obstruction, it is necessary to free the bowel at an opportune time.

Somewhat more than a year ago I did the thirty-first operation for a girl who had been operated on thirty times previously for obstruction of the bowel and fistula. The entire bowel was freed, from the duodenojejunal angle to the cecum, and on completion of the operation air was injected into the peritoneal cavity. The patient's sacrum was kept elevated with three pillows so that the air would accumulate beneath the anterior abdominal wall, chiefly about the umbilicus, and mechanically separate bowel and anterior abdominal wall during the healing phase. This patient has remained quite well without pain or complaint referable to the bowel, except for the occurrence of slight vomiting during a recent attack of salpingitis of the fallopian tubes. Air injected in this manner is not likely to be of any value in the prevention of interintestinal adhesions. The patient's own serum (defibrinated blood) should prove to be fully as efficacious as, and have some distinct advantages over, amniotic fluid for this purpose.

7. In the use of suction with the nasal catheter after abdominal operations, patients are permitted to drink clear fluids freely from the start. When an actual mechanical block exists in the bowel, however, it has been thought wise to deny the patient fluid by mouth until the intestinal distention is gone. A more accurate estimate of the fluid and gas removed is also obtained by this precaution.

Miss G. R. was admitted to the University hospital on March 20, 1932, with an acute intestinal obstruction three months after operation for acute suppurative appendicitis elsewhere. At the time her appendix was removed it had been necessary for her to remain in the hospital four weeks because of profuse drainage from her wound. On her admission to the University Hospital, twenty-four hours after the onset of vomiting, she complained of intermittent cramplike pain occurring at frequent intervals. There was no abdominal tenderness, and the abdominal wound was healed. Stethoscopic examination revealed loud intestinal noises at the acme of the pain, establishing the presence of intestinal colic. Rectal examination disclosed a small tender mass in the pelvis (culdesac abscess). A film of the abdomen (fig. 3) showed moderate distention of several coils of small intestine. The presence of gas in the colon following expulsion of gas with previous enemas which had been administered on admission indicated that the obstruction was incomplete. An administered Noble's enema [7a] was returned with some flatus but no relief of pain, and suction by nasal catheter was instituted. There was immediate cessation of pain after the insertion of the catheter, and the tube quickly found its way into the duodenum, as shown by a subsequent plate. The decompression progressed satisfactorily, and after two days' suction the gaseous shadows were scarcely visible on the x-ray film. On the third day the catheter was clamped occasionally, and clear liquid was permitted by mouth. Suction through the nasal catheter was continued intermittently until the fifth day after admission, since which time there had been no recurrence of pain.

During the first night (twelve hours), 900 cc. of fluid and 200 cc. of gas were aspirated through the nasal catheter. In the next twenty-four hours, 800 cc. of fluid and 600 cc. of gas were obtained with the nasal catheter and 1,950 cc. of gas and 50 cc. of fluid by the rectal suction. In the following twenty-four hour period, 450 cc. of fluid and 800 cc. of gas were aspirated by the nasal catheter and 100 cc. of gas and no fluid by a rectal tube similarly connected with a suction apparatus. A Noble's enema was given with effectual results and rectal suction was discontinued. In the following twenty-four hours, 1,200 cc. of fluid and 900 cc. of gas were obtained by the nasal catheter and in the last twenty-four hour period before suction was abandoned, 400 cc. of fluid and 700 cc. of gas were removed.

The worth of decompression by nasal suction was recently put to a severe test in the instance of a man of 35 in whom acute intestinal obstruction developed fourteen days after operation for suppurative appendicitis.

A. W., aged 35, had complained of intermittent "gas pains" for about thirty hours before vomiting occurred. Attempts at obtaining expulsion of gas with enemas had proved ineffectual. A diagnosis of acute intestinal obstruction was not established until vomiting took place. At this time (Feb. 26, 1932, 1:45 a. m.) considerable distention of several coils of small intestine in the left half of the abdomen was apparent on the x-ray film (fig. 7 *A*). Suction siphonage by nasal catheter was started at once with immediate cessation of cramps, and after a few hours the patient declared himself to be comfortable and stated that he had never felt better in his life. An expression that he frequently repeated during the period that the suction was employed was, "I feel like a million dollars."

Bedside films were made again at 2 p. m. and 11 p. m. (fig. 7 *B*). These showed the stomach emptied, but only a slight diminution in intestinal distention had occurred. On the evening of February 26, the suction was increased to about 250

7a. Noble's enema: turpentine, 1 drachm (3.75 cc.); glycerin, 2 ounces (59.2 cc); magnesium sulphate, 3 ounces (89 cc.); water, 4 ounces (118.4 cc.).

cm. of water (the nasal suction employed being 75 cm.), but when the film was made at 11 o'clock, it was not noted that the distal end of the catheter had returned into the lower end of the esophagus. This was not noted until 7 a. m. on February 27. Only 700 cc. of gas and 800 cc. of fluid had been aspirated since the institution of suction.

The tube was pulled out again and reinserted and apparently soon made its way into the duodenum; the decompression of the distended bowel progressed nicely, as shown in a film made at 9 p. m. on February 27 (fig. 7 *C*). When the drainage of gas and fluid was computed at 6 a. m. on February 28, it was found that 1,100 cc. of gas and 1,900 cc. of fluid had been aspirated during the preceding twenty-four hours. An x-ray film taken at 8 a. m. showed the small intestine to be absolutely decompressed, no gaseous distention being present. At 10:00 a. m., the suction was discontinued by clamping the tube, and 1,250 cc. of clear strained liquids was allowed by mouth. At 6:30 p. m. an enema was given, following which some flatus was expelled. At 7:00 p. m. another bedside film was taken (fig. 7 *D*). Gas was noted in the colon for the first time since the diagnosis of obstruction had been made, indicating that the continuity of the bowel was being reestablished. Incident to the allowance of oral fluids and temporary discontinuance of suction, however, gas had again accumulated in the small intestine. At 8:15 p. m. suction was commenced again. Suction was also applied to a rectal tube, and during the night 100 cc. of gas and 100 cc. of fluid were aspirated by the nasal tube and 100 cc. of gas and no fluid by the rectal tube.

An x-ray film taken at 8:00 a. m. on February 29 showed much less gas in the bowel than twelve hours before, and clear fluids were again permitted by mouth. About 10:00 a. m. it was noted that gas began to collect rapidly in the bottle connected with the rectal tube. When the computation was made at 6:00 a. m. on March 1, it was noted that the following drainage had occurred in the preceding twenty-four hour period: by nasal catheter, 1,800 cc. gas and 2,810 cc. fluid; by rectal tube, 7,700 cc. gas and no fluid. Suction was continued intermittently through March 1, and by March 2, the patient was passing gas freely by rectum and complained of being extremely hungry. Nasal suction was discontinued, and fluid and solid food were permitted by mouth. The continuity of the intestinal lumen remained patent despite the administration of food, and it was manifest that there had been complete recovery from the obstruction of the bowel.

In the last few days of the period of intestinal obstruction it was apparent that a small culdesac abscess was forming. The patient evacuated mucus several times a day by rectum without expelling gas or feces. Following the discontinuance of rectal suction, several warm enemas a day were given, and by the time that the patient was ready to be up no trace of the abscess could be made out on digital examination of the rectum. There has been no recurrence of "gas pains" since dismissal from the hospital.

Though the decompression of the distended small bowel progressed fairly rapidly in this instance, it was the appearance of gas in the colon (fig. 7 *D*) that afforded encouragement in knowing that the obstruction had been overcome. Visualization of gas in the colon in complete obstructions is synonymous with the reestablishment of the intestinal current and has in a manner the same significance for patient and physician as the exultation and delight experienced by a shipwrecked mariner on the sight of another sail.

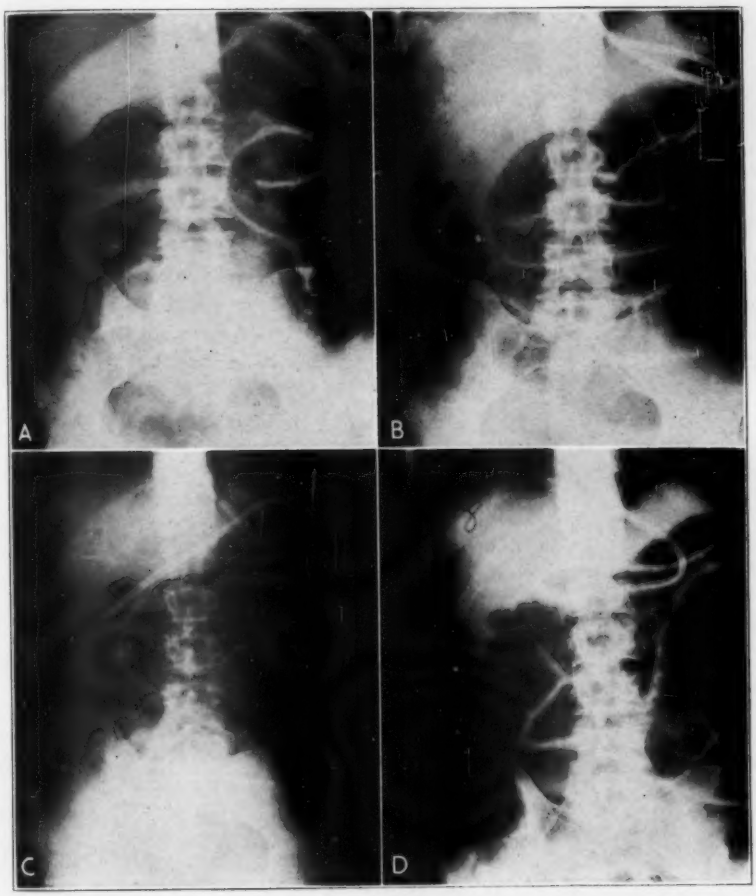

Fig. 7.—Roentgenograms of the abdomen of Mr. A. W., 35 years old. *A* was taken fourteen days after operation for acute suppurative appendicitis. There is considerable distention of the stomach and the small intestine. *B* was taken at 2 p. m., thirteen hours after the institution of suction. The stomach is empty, but there has not been much reduction of the distended coils of the small intestine. *C* was taken forty-four hours after suction was started. An effectual reduction in distention of the small intestine is present, but practically no gas has progressed into the colon. *D* was taken twenty-two hours later. There is now considerable gas in the colon, heralding the establishment of the continuity of the intestine.

Mr. J. E., aged 50, was admitted to the hospital on April 7, 1932, complaining of intermittent crampy pain, nausea and vomiting, of forty hours' duration. On admission paroxysms of pain occurred at intervals of from five to ten minutes, and loud intestinal borborygmi were heard with the stethoscope at the acme of the pain. The patient had also noted the concomitant occurrence of noise and pain. There was slight but definite tenderness of the abdominal wall, most easily demonstrated as rebound tenderness. One month previously the patient had a lesser attack of pain accompanied by nausea and vomiting from which he spontaneously recovered. He had had no previous abdominal operation. For ten years he has had a right inguinal hernia and has worn a truss for five years. The hernia has not recently been incarcerated. X-ray films of the abdomen revealed considerable distention of the upper coils of small intestine. Gas and feces were evacuated with enemas, but the pain still continued.

The slight tenderness and rigidity present here indicated that there had been an escape of fluid into the peritoneal cavity, suggesting the presence of a potential strangulation mechanism. As the pulse and temperature were not appreciably elevated, I thought that one might proceed cautiously with suction, watching the patient carefully for signs of increasing tenderness. Suction by nasal catheter was started at midnight on April 7. There was also immediate relief of pain, though the tenderness was still definitely demonstrable. On April 8, at 6 a. m., 800 cc. of gas and 600 cc. of fluid had been aspirated. There was not much reduction in the intestinal distention, as shown in the x-ray film. An enema was given during the night without effect. Two capsules of amyl nitrite were broken in some gauze for the patient to inhale, and he was turned on his right side. Some time later the tube made its way into the duodenum, and the suction became more effective. During the next twenty-four hours (April 9 at 6 a. m.), 1,700 cc. of gas and 900 cc. of fluid were aspirated, and an x-ray film of the abdomen showed definite reduction in distention, though the upper jejunal coils were still somewhat dilated. During sleep at 1 a. m., April 10, the patient pulled the catheter out. A Rehfuss tube was then put down through the mouth. No reliable measurement of gas could be made for the preceding twenty-four hours because of the extraction of the tube by the patient. An x-ray film made on April 10 at 8 a. m. showed almost complete disappearance of gaseous distention of the jejunum, and gas was present in the colon.

Clear fluids were permitted in small amounts by mouth, and the catheter was occasionally clamped. Suction was discontinued on April 12, and a roentgenogram taken on April 13, following a barium sulphate meal, showed the motor transit of barium through the bowel to be normal. The patient was allowed to be up and about, but wished to have his hernia repaired, which was done on April 25. Because of the antecedent obstruction, a low rectus incision was made permitting of examination of the abdominal contents as well as repair of the sac from within. A long strand of omentum was found to be engaged and adherent in the mouth of the hernial sac. It was delivered, and the hernial opening was closed. About 3 feet proximal to the cecum, there was an area about 2 inches in length where the small intestine presented a circular, thickened, opaque whitish plaque, representing undoubtedly the site at which the obstruction had occurred. The patient convalesced uneventfully and was dismissed on May 4.

R. B., a boy, aged 8 years, was seen in consultation at the Norwegian Deaconess Hospital with Dr. Frank Anderson, through whose courtesy I am permitted to include this case report. This patient had been admitted on Feb. 26, 1932, complaining of pain, nausea and vomiting. A diagnosis of intussusception was made,

and at operation an enteric intussusception of about 6 inches in length was reduced. The postoperative convalescence was uneventful, and the patient was dismissed on March 11.

On March 18, he was readmitted because of recurrence of his complaint. At operation a reinvagination of the small intestine was found to have occurred. Reduction could not be effected, and the bowel was of questioned viability. Resection was therefore done, and an end-to-end anastomosis made. There was gradual improvement until March 22, when the patient began to complain of abdominal cramps, followed by nausea and vomiting, and these were the complaints at the time I saw the boy on the evening of April 4.

Intermittent colic occurred periodically, and coincident with the pain loud intestinal borborygmi could be heard with the stethoscope. There was moderate distention of the abdomen, which was tympanitic above and somewhat dull immediately below the umbilicus, where the margins of the wound were indurated. The remainder of the abdomen was rather soft, though slight rebound tenderness could be elicited. From the indurated portion of the wound a fecal-smelling exudate could be expressed (colon infection, not feces). An x-ray plate made of the abdomen showed moderate distention of the small intestine. The button used for anastomosis was seen to be in the region of the ileocecal valve.

Suction siphonage by nasal catheter was instituted with immediate relief of pain and distress. Para-oral fluids were freely administered. During the next forty-eight hours, 1,500 cc. of gas and 1,460 cc. of fluid were aspirated. Some gas was evacuated by rectum, and the patient was permitted a little water and clear fluid by mouth, the tube being clamped occasionally. During the next twenty-four hours, 500 cc. of gas and 300 cc. of fluid were aspirated, and in the following twenty-four hour interval, 600 cc. of gas and 200 cc. of fluid; the button was expelled on April 9. The suction was continued intermittently for the next three days, approximately 600 cc. of gas and 300 cc. of fluid being aspirated each day. The patient was discharged well on April 18.

Mr. B. V., a hemophiliac, aged 23, was admitted to the hospital on May 7, 1932, because of abdominal pain, nausea and vomiting. The pain had commenced on May 3; on May 4 it increased, and in the evening he vomited. He continued to take liquid food, but on May 5, the pain, nausea and vomiting were greatly exaggerated. On May 6, following a dose of salts, a small tarry stool was passed; the pains became more severe, and the vomiting which was previously bilious became brown.

His physician, Dr. C. Roholt of Waverly, Minn., saw the patient at this time and observed tenderness in the lower left quadrant of the abdomen as well as a tender oblong mass at the same site. On May 7, there was tenderness in both lower quadrants. An enema returned a bloody stool. The vomiting was persistent and frequent, and borborygmi were audible to the unaided ear at the acme of the pain.

The patient had previously been admitted to the University Hospital because of a hemorrhage into the knee joint following slight trauma and a spontaneous hemorrhage into the right eye in January, 1930. The patient has suffered from prolonged bleeding since infancy. One brother died in infancy of hemorrhage from a slight cut in the tongue. A maternal uncle has hemophilia. On examination the patient was found to be blind in the right eye. The blood pressure was 134 systolic and 88 diastolic. The hemoglobin was 72 per cent and the white blood cells, 14,190. The clotting time was two hours and twenty minutes. The urine contained considerable albumin as well as many red blood cells, hyaline and granular casts. There was moderate distention of the abdomen with slight tenderness

and rigidity of the entire abdomen, more marked below. At the height of the intermittent pains which recurred periodically, intestinal borborygmi were plainly audible. An x-ray film taken of the abdomen showed considerable distention of the upper loops of jejunum. A slight amount of gas appeared to be present in the colon.

A diagnosis of hematoma in the bowel and mesentery causing mechanical obstruction was made and suction was instituted, liberal amounts of fluid being given para-orally. Considerable bloody fluid was evacuated from the stomach at first. On the morning of May 8, the patient felt much better; 2,200 cc. of gas and 1,000 cc. of fluid had been aspirated during the previous night. Two hundred units of theelin were given by the medical staff, and twenty minutes later the coagulation time was five minutes. An x-ray film made at 1 p. m. on May 8 showed marked reduction in the intestinal distention. Gas was evacuated by rectum. On May 9, the patient appeared much improved and had no complaints. Only 400 cc. of gas and 300 cc. of fluid having been aspirated in the previous twenty-four hour period, suction was discontinued and the patient was permitted fluid by mouth. The clotting time at 2 p. m. was twenty minutes. Fifty units of theelin was given on May 10, the obstruction having cleared up. The patient was transferred to the medical service for continuance of the treatment of hemophilia and has since been dismissed.

Suction by nasal catheter alone proved unsuccessful in the case of a boy of 16 years convalescing from an operation for acute suppurative appendicitis.

J., aged 16, was admitted to the hospital on Oct. 28, 1931, for an appendectomy. On the fourth day after operation symptoms of obstruction developed. Pain continued despite effectual evacuations with enemas, and suction was instituted. Pain ceased at once, but successive roentgenograms (fig. 8 *A* and *B*) made at an interval of twelve hours failed to show any appreciable decrease in intestinal distention. An unsuccessful attempt was made to pass the nasal tube into the duodenum under the fluoroscope in this case, it being felt that spasm of the pylorus was probably responsible for the failure. Enterostomy was then done, following which a decrease in the intestinal distention and progress of gas into the colon became apparent (fig. 8 *C*). The patient was discharged well thirty days after admission.

This was the third case in which suction had been employed in the treatment of acute intestinal obstruction, and in the first two instances the decompression progressed rapidly. I was a bit dismayed by the persistence of the distention in the small intestine and felt that enterostomy, an operation of established value, should take precedence over this unorthodox procedure of emptying the bowel. The patient was comfortable from the time that suction was started, however. With the experience gained in similar stubborn instances, previously related, I should now have tried somewhat longer before having recourse to enterostomy. In the rather limited experience which opportunity has afforded for trial of the method, I am inclined to believe that in practically all instances of obstruction of the small bowel decompression can be done by this method if the tube can be gotten through the pylorus when decompression cannot be performed with the catheter in the

stomach.[7b] In those instances, however, in which the obstructive mechanism does not relent, resort must be had to operation as the following case will illustrate.

R. E., a boy, aged 8 years, was admitted to the University Hospital on March 20, 1932, with a diagnosis of acute appendicitis. The symptoms were of seventy-two hours' duration. The temperature on admission was 102 F., and the pulse was 110. The white blood cells were 23,850, 90 per cent of which were polymorphonuclears. There were diffuse abdominal tenderness and rigidity. No masses were palpated by rectum, though the tenderness was marked.

A diagnosis of diffuse peritonitis of appendical origin was made, and the conservative plan of treatment which had been employed in such instances was carried out, viz., massive hot packs to the abdomen, para-oral fluids and suction by nasal catheter to relieve distention. The patient had a fairly stormy time, but gradually improved and appeared to do well after March 29.

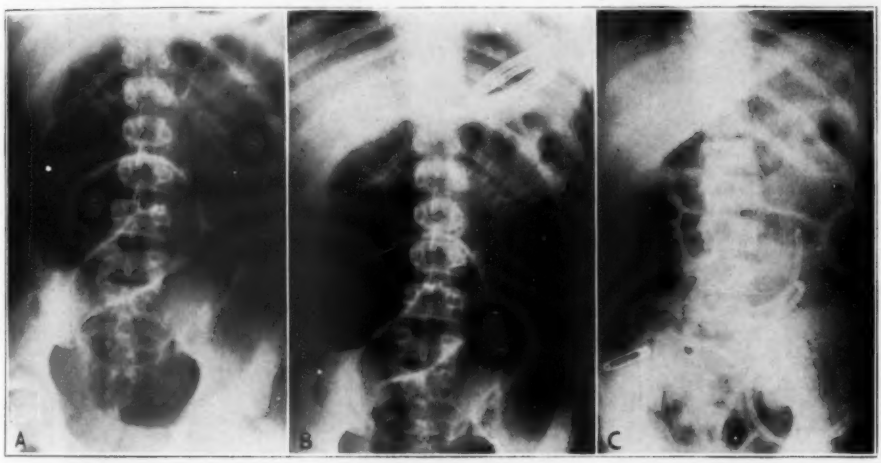

Fig. 8.—Roentgenograms of the abdomen of a boy, aged 16. *A* shows acute obstruction which developed four days after appendectomy. *B* was taken after suction siphonage by nasal catheter had been employed twelve hours; there is no appreciable reduction in distention; *C* was taken twelve hours after enterostomy; gas is present in the colon and the distention of the small intestine is considerably diminished.

On April 23, at which time the patient appeared to be otherwise quite well, intermittent pain occurred. Gas was expelled with enemas, but the pain continued. On the morning of April 24 the cramps were severe, and loud borborygmi were

7b. It was soon learned that it is an important item in accomplishing decompression with the duodenal catheter to have holes cut back on the tube for about 10 inches (25.4 cm.), so that constant suction may be exerted in the stomach as well as in the upper reaches of the small intestine after the tip of the catheter has gone beyond the pylorus. The details of technic of decompression have been described by Dr. J. R. Paine and myself elsewhere (Nasal Catheter Suction Siphonage: Its Uses and the Technic of Its Employment, Minnesota Med. **16**:96 (Feb.) 1933.

audible with the stethoscope at the acme of the pain. Considerable distention was present, and when vomiting occurred at 2 p. m., a roentgenogram was made of the abdomen which showed considerable distention of both large and small bowel to be present (fig. 9 *A*). Masses of feces are visible in the ascending colon on the film. Though lumps of hard feces were expelled with enemas, the pain continued.

A diagnosis of partial obstruction was made (gas in colon), and suction by nasal catheter was commenced; pain stopped at once. Fluid was interdicted by mouth, but was administered intravenously. A roentgenogram made at 9: 30 p. m. showed some decrease in distention of the upper coils of the small intestine; 3,625 cc. of gas and 150 cc. of fluid had been aspirated in the meantime. An x-ray film made at 10: 30 a. m. on April 25 showed considerable reduction in the gaseous distention of the small intestine with almost complete evacuation of the gas in the colon (fig. 9 *B*). An abscess on the external aspect of the right thigh containing considerable greenish pus (staphylococcus) was opened at this time. On April 26, the patient was permitted fluid by mouth, the catheter being clamped periodically.

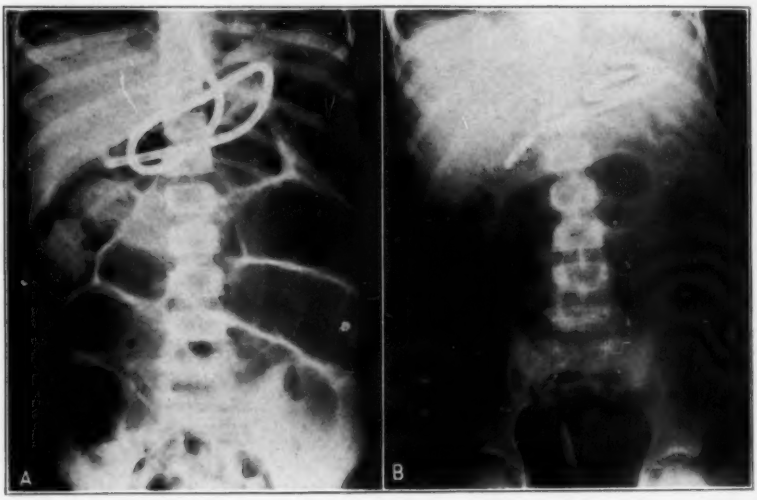

Fig. 9.—Roentgenograms of the abdomen of a boy, aged 8 years. *A* shows acute obstruction; about five weeks previously he was admitted to the hospital with diffuse peritonitis of appendical origin which subsided under conservative treatment. *B* was taken twenty hours after instituting suction. (The obstruction developed again after appendectomy, and the bowel was again decompressed by suction siphonage with a nasal catheter, but when feeding was started gaseous distention recurred necessitating enterostomy. Later, release of the constricting agent also became necessary.)

On April 27, the suction was discontinued, and the patient continued to do well. On the afternoon of April 29, pain was complained of in the right lower quadrant, and there was definite rebound tenderness. An x-ray film showed some return of gaseous distention of the small intestine. Suction was recommenced and carried through the night. The next morning, appendectomy and drainage of an appendical abscess was done. The patient had already been ill for more than a month, and transfusions had meanwhile been given. Nasal suction was continued intermittently for three days after operation. By May 3, the patient was doing very well, taking solid food and exhibiting daily improvement. On May 8, feces made their

appearance in the wound (fistula at site of removal of appendix), but there were no complaints, however. On May 10, the patient again complained of abdominal pain attended by borborygmi, and there was emesis of 200 cc. The abdomen was distended, and enemas gave no relief. An x-ray film demonstrated considerable distention of the small intestine.

Suction was begun with relief of pain and reduction of the distention, as visualized on the x-ray film. The nutritional problem in this child who had been ill for seven weeks was now a significant consideration. Every attempt to feed him, however, resulted in an increase of the intestinal distention. An enterostomy was done under local infiltration anesthesia on May 16 without reaction. At operation strands of fibrin were visible over the coils of bowel, undoubtedly a residual of the diffuse peritonitis for which the patient was admitted. Suction was subsequently applied to the catheter in the bowel. By May 18 there was no distention, and the patient declared himself to be hungry and began to take solid food. A few days later symptoms of obstruction developed again despite the fact that the enterostomy tube was draining freely. A direct attack [8] was then made on the obstructing mechanism near the site of appendectomy, and a catheter was inserted into the fistulous opening in the cecum. The patient has been improving but will probably need an operative procedure to close the fistulous opening in the cecum persisting after appendectomy.[8a]

Satisfactory decompression was obtained following the first attack of obstruction of the bowel in this child. Shortly after appendectomy and drainage of an appendical abscess, obstruction recurred. The small bowel could again be satisfactorily decompressed, but attempts at feeding resulted in increased distention, indicating the necessity for enterostomy. Later release of the persistent obstruction bands also became necessary.

This series of eleven cases in which decompression was achieved by suction through a nasal catheter (enterostomy was also done in two) is marred through death in one case (Mr. O. N., aged 52), for which the method is in no way to blame. A successful decompression of the small intestine was obtained by suction, but the obstruction continued. Because of the long continuance of the obstruction and the weakened condition of the patient, repeated efforts were made to interrupt the suction and allow liquid nourishment orally. Following each attempt the small bowel distended again, and operation was undertaken. Whereas, ordinarily in such cases I have done enterostomy as a routine measure, in this instance, because of the preliminary decompression and because of an unwarranted interest as to the nature of the obstruction (my impression was carcinoma of the small intestine), I took the liberty of locating its site. A small mass was found in the terminal ileum, apparently confirming the impression of carcinoma. A gentle attempt was made to elevate the mass, and directly an escape of fecal material into the wound from the mass was observed; the patient's fate was sealed by this unhappy maneuver. The leakage was mopped up, the

8. In a fairly large number of enterostomies which I have performed this is the second instance in which a subsequent operation on the site of obstruction was necessary in order to establish the continuity of the intestine.

8a. The fistulous opening in the cecum has since been closed, and the patient in a recent letter declared himself to be entirely well.

tiny opening in the bowel closed and a proximal enterostomy done, but these were futile corrective measures in the presence of this spillage, and the patient died of peritonitis seven days later.

The patient's history anterior to operation was briefly as follows:

About eight weeks before entry to the hospital the patient was seized with abdominal pain followed by hiccup and frequent vomiting. There had been blood in the stool and a loss of 30 pounds (13.6 Kg.) in weight. On examination the patient was found to be much dehydrated and showed evidence of considerable recent loss of weight. Visible and palpable peristalsis was present, and loud intestinal borborygmi were audible synchronous with the crampy pains of which the patient complained. The blood urea was 51 mg. per hundred cubic centimeters; blood chlorides, 395 mg., and the result of the Van Slyke test was 76 per cent by volume. The hemoglobin was 108 per cent. Moderate distention of the small intestine, especially in the left portion of the abdomen, was present on the x-ray film. Suction siphonage by nasal catheter was continued for the next three days before the bowel showed disappearance of the gaseous shadow in the small intestine. Each attempt at interrupting suction in order to feed the patient orally resulted in increasing distention. On the sixth day after admission, because of the conjectured nature of the obstruction, operation was done; 11,050 cc. of fluid and 4,800 cc. of gas had been aspirated during the interval.

Postmortem examination failed to disclose the cause of the obstruction. The most logical explanation would appear to be that a foreign body (not discovered at operation or autopsy) perforated the bowel and gave rise to an inflammatory mass, the manipulation of which caused the leakage.

Simple enterostomy would have obviated this unhappy issue. Unfortunately, however, too often, an error committed cannot at once be undone and another plan projected without accepting some penalty from the indiscretion. Experience in surgery would command no special value could one, as in O. Henry's "Roads of Destiny," on following out unsuccessfully one course of action start on another untrammeled and with peace of mind and without handicap seize on another mode of attack. A lesson taught here should be that the safest procedure is the most conservative. Suppression of curiosity as to the nature of the obstruction would have saved this patient's life and me considerable embarrassment.

Suction siphonage by nasal catheter has its shortcomings and is not to be recommended in the treatment of strangulation obstruction, which is to be recognized clinically in the occurrence of rebound tenderness.[9] In carcinoma of the sigmoid flexure in which gaseous distention involving only the colon usually obtains, due to competence of the proximal ileocecal valve, drainage of the upper reaches of the bowel by catheter would be inadequate; and when the cause continues to operate, as in

9. Doubt may be entertained by some as to whether simple and strangulation obstructions can be differentiated without operation. In any borderline case certainly operation is to be done, but I feel definitely as set forth elsewhere (Northwest Med. **30**:389 [Sept.] 1931) that the physical findings will serve to distinguish these varieties.

the instance of the first patient previously referred to, it is not to be expected that drainage through a nasal catheter alone will suffice. In adhesive types of obstruction in which the cause usually ceases to operate following decompression of the bowel, the method should have its greatest usefulness, and in postoperative obstruction particularly the method should prove to be an agent of therapeutic value. In the use of the method it is wise to follow the intestinal decompression by taking frequent x-ray films of the abdomen (portable) until it is obviously apparent, from the degree of intestinal distention present, whether the method will be successful or not.

It is extremely important that enough fluid be administered by the subcutaneous and intravenous routes during the time that suction is in force to insure a good daily urinary output (about 1,000 cc.). If the distention subsides satisfactorily and a liberal urinary output obtains, it is not necessary to follow the chemical changes in the blood. Except in unusual instances the daily output of urine is as reliable an index of whether enough fluid and salt are given as are frequent determinations of the blood chlorides, urea and carbon dioxide-combining power of the blood. In the cases referred to in this paper, from 4,000 to 6,000 cc. of fluid has been given daily. With the removal of fluid from the stomach and upper reaches of the bowel it is imperative to give considerably more fluid than the ordinary patient requires. Physiologic solution of sodium chloride is usually given subcutaneously on the outer aspects of the thigh beneath the fascia lata and 10 per cent dextrose solution (sometimes in physiologic solution of sodium chloride) intravenously.

Measures to assuage pain have been unnecessary. With the institution of suction the pain almost invariably miraculously ceases. An occasional lesser cramp is sometimes complained of. In the latter cases in this group, most of them instances of recent postoperative obstruction following surgical intervention for suppurative conditions, I have employed hot packs to the abdomen as a routine measure. This helps to relax the abdominal muscles and is worth while.

With the oral source for increase in the gaseous distention shut off by suction, even in those instances in which the decompression of the distended bowel has progressed slowly, it has been remarkable how the patient's condition, as well as his comfort, improved incident to the interruption of accretion in distention. Borborygmi may be made out with the stethoscope, but they are not as violent and as frequent as before. When the bowel has become accommodated to a certain grade of distention, if no tendency toward progression is exhibited, the peristaltic activity apparently lessens. A few of the patients have complained a little about the presence of the tube in the throat, but the instillation of a few drops of nasal oil has usually mitigated this distress.

In a few patients the end of the tube persisted in the stomach throughout, and a satisfactory evacuation of the small bowel was

obtained. Where the decompression has progressed rather slowly, I have been anxious to see the catheter make its way beyond the pyloric sphincter into the duodenum. In one instance (an early case), previously listed, the decompression failed because it was impossible to get the catheter into the duodenum. In two subsequent cases, the inhalation of amyl nitrite appeared to facilitate the entry of the catheter into the duodenum.

There is one distinct advantage that enterostomy presents over decompression by means of suction through a nasal catheter, viz., the opportunity to feed the patient as soon as the decompression has been effected. In complete types of simple obstruction it may take some time for the continuity of the bowel to become reestablished, as heralded by the appearance of gas in the colon despite complete disappearance of the distention. Fortunately, in most instances, successful decompression is followed quickly by automatic reestablishment of the continuity of the bowel, but in those in which an interval obtains between these events, attempts at feeding without an enterostomy usually result in a renewal of the distention.

The rationale of the success of this method in decompressing the bowel undoubtedly resides in the fact that the chief source of the gaseous distention is swallowed air. When the decompression is established by enterostomy, there is frequently little escape of gas or fluid after the initial release of the catheter. The escape of some gas and fluid permits the angulation or kinking of the bowel to become adjusted and righted, and the continuity of the intestinal current becomes automatically established. This explanation no doubt holds true in some measure of the decompression effected by nasal suction. The removal of intestinal gas through the nasal catheter decreases the distention of the obstructed bowel and permits the gas to go down into the lower reaches of the bowel.

All persons with simple acute mechanical intestinal obstruction observed at the University Hospital since August, 1931, when the method was first successfully employed, with the exception of three with carcinoma of the sigmoid flexure, which, as previously stated, constitutes a contraindication to its use, have been treated by suction siphonage through the nasal catheter. During this interval (until May 1, 1932) only twelve such patients have been seen, all of whom are referred to in this report. Decompression was unsuccessful in an early case by this method because the catheter did not enter the duodenum. In another, decompression was successfully effected by suction but recurred sixteen days later. The distention of the small intestine was again satisfactorily dealt with by suction by nasal catheter, but attempts at feeding the child who had been ill for more than seven weeks resulted in increasing distention. Enterostomy was done to permit of feeding. In nine instances a satisfactory decompression was obtained by suction

alone. In another, also decompressed by suction, in which the obstruction continued, the patient unfortunately died following an ill advised attempt to ascertain the nature of the obstruction and reestablish the continuity of the bowel.

The method has also been successfully used in a fairly large number of instances of subacute and chronic obstructions not enumerated in this report, due chiefly to narrowing of the pelvic colon and rectum by carcinoma of the pelvic genital organs of the female. Through relief of the obstruction by nasal and rectal suction in such instances and subsequent regulation of the diet and administration of liquid petrolatum, many such patients have been spared a terminal colostomy. Suction by nasal catheter has been employed also as an auxiliary aid in the relief of distention following the release of strangulation obstructions (hernias and intussusceptions).

NOTE.—At the time of proof-reading this paper, twenty-four cases of acute mechanical obstruction of the small intestine have come under observation since suction siphonage by nasal catheter was first employed in the treatment of mechanical obstruction. Decompression has been done in twenty of these by suction siphonage through nasal catheter alone. In four instances enterostomy also became necessary. In the entire group there have been two deaths (8.3 per cent): the one referred to in the text of this paper and another, the case record of which will be briefly reviewed. Mrs. H., aged 66, was admitted to the University Hospital on Aug. 10, 1932, for disease of the gallbladder following a severe attack of a few weeks previously. In addition to a well developed acute intestinal obstruction, the cause of which was a packet of gallstones resembling the cast of a gallbladder impacted in the ileum, she had a large infected bed sore over the sacrum, with considerable fever. Suction siphonage by nasal catheter was instituted, and the gaseous distention of the small bowel disappeared. Owing to her generally poor condition, feeding was attempted through the catheter, and alternate feeding and suction proved successful despite the continued presence of the gallstones in the small intestine. Six weeks later signs of acute intestinal obstruction again developed. Pituitary extract was unfortunately given; perforation of the intestine occurred, and the patient died of peritonitis a few days later. A timely operation in the unobstructed phase would perhaps have avoided this disaster.

A questionnaire has been submitted to the patients treated in this manner, and the majority state that they have no complaints referable to the intestinal canal. This material together with other details of treatment will be made the subject of a subsequent report.

OPERATION

Elsewhere [10] I have described in some detail the conduct of the operative procedure in acute intestinal obstruction. Here the principles of operation will alone be briefly outlined.

Anesthesia.—In patients with early intestinal obstruction the choice of the anesthetic agent is not a matter of paramount importance. Patients with late obstruction exhibiting considerable regurgitant vomit-

10. Wangensteen, O. H.: The Diagnosis and Treatment of Acute Intestinal Obstruction, Northwest Med. **30**:389 (Sept.) 1931.

ing are poor risks for an inhalation anesthetic because of the danger of aspiration. I have used spinal anesthesia almost entirely, with complete satisfaction. In postoperative obstructions, local infiltration is usually adequate. In infants, local infiltration reenforced by a small amount of ether serves the purpose well.

Incision.—When the type and location of the obstruction remain undetermined, a subumbilical midline incision is best made. Care must be taken that the urinary bladder at operation is empty, especially following the generous preoperative administration of fluid. In the relief of postoperative adhesive obstruction by enterostomy, a small oblique incision opposite the umbilicus over the outer edge of the left rectus muscle has been made as a routine procedure.

Choice of Procedure.—In the operative relief of simple obstruction the surgeon should permit himself to be guided solely by the general condition of the patient. In early cases, the operation of election can be done, viz., locating the exact site of obstruction, determining its nature and releasing the block that obstructed the bowel. In late cases, enterostomy, an operation of necessity, is much the safer procedure. All anastomotic procedures on the dilated intestine are to be deliberately avoided.[11] In strangulation obstruction, the condition of the bowel after its release and not the patient's general condition must determine the choice of procedure. No matter how ill the patient, if nonviable bowel is present, it must be excised. Exteriorization with the establishment of complete external fistula followed by a secondary anastomosis carries a much lesser risk to the patient's life than excision and primary anastomosis.

Enterostomy.—Site: The nearer the enterostomy is to the point of obstruction, the more efficient is the drainage. A deliberate attempt to make a "high" enterostomy is to be avoided. I have observed that the amount of drainage (gas and fluid) is correlated only with the degree of obstruction present, and not with the level at which the enterostomy is done.

Technic: A distended coil of bowel is seized and drawn out through the wound. Should the bowel present with any difficulty, the incision should be adequately enlarged to avoid injury to the serous coat of the distended intestine. Before placing a single stitch in the distended wall of the bowel, it is highly essential that the segment selected be emptied of its content. This may be done by milking it out between the fingers and isolating the collapsed segment between clamps; or if the wall of the bowel is too tense, the rubber-covered clamps should be first applied and the distended segment aspirated with a long fine needle and a large syringe.

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11. Congenital intestinal atresia is an exception to this rule, the only reported recoveries having occurred after an entero-anastomosis. Infants do not tolerate fistula of the small intestine.

Immediately following emptying of the thin dilated bowel, the circular muscle of the intestine contracts and the wall of the bowel presents a surprising increase in thickness, rendering suture of the bowel a safe procedure and precluding leakage from the stitch holes which would otherwise occur. Removal of the content from the segment on which enterostomy is to be performed also does away with all danger of the slightest spillage on introduction of the catheter into the bowel.

In the performance of enterostomy for acute intestinal obstruction, there must be absolutely no spillage. The slightest soiling invites peritonitis and is synonymous with failure. The needles and suture material should be fine (chromic catgut 000 or silk). A urethral catheter, no. 14 French, is scored as shown in the accompanying diagram (fig. 10) and a fine 000 chromic catgut ligature is fixed in the groove. The catheter is then buried in the wall of the bowel by the Witzel technic, the suture starting about 1½ inches proximal to the scoring on the catheter. The proximal end of the catheter must always be clamped off before the bowel is punctured. A Bard-Parker blade no. 11 or a fine cataract knife is used for the puncture. The distal end of the catheter is pushed into the lumen of the bowel, and the enfolding stitch is continued for another 1½ inches. An aseptic enterostomy can be done in this manner (fig. 11), the most important feature in establishing an effectual decompression with minimal risk.

The segment on which enterostomy has been performed can be safely dropped back into the peritoneal cavity without the slightest fear of leakage. Omentum need not be sought to cover it.[12] When the catheter comes away about the tenth to twelfth day (unless adhesive fixation on the skin continues to retain it), the tiny peritoneal tunnel closes spontaneously without subsequent leakage. Persistent fistula after removal of the catheter is due to employment of too large a catheter or to the use of the multiple purse string suture which angulates the bowel and obstructs it. A no. 14 catheter will drain the bowel as well as a garden hose.

Exteriorization.—The time factor and the rigidity of the constricting agent are the significant items in determining whether a strangulated bowel will be found to be nonviable. Whereas many types of obstruction of the bowel are potentially instances of strangulation obstruction from the beginning, primary thrombosis or embolism of the mesenteric vessels is the only type of strangulation in which excision of the devitalized bowel is mandatory. In all other potential strangulations, release of the obstruction before the anatomic changes consequent on prolonged interference with the blood supply of the segment have occurred obviates the necessity of dealing with a nonviable bowel. A fine balance of judgment must occasionally be exercised in dealing with a questionably viable bowel in a patient in poor condition. Should the decision be made to leave it, the nutrition of the segment should be protected by making a proximal enterostomy to prevent distention, or the same effect can be insured by the use of suction siphonage by nasal catheter after operation. Bowel that is frankly nonviable should be excised. A resection with immediate anastomosis is to be deliberately avoided. Even though

12. When performing enterostomy for postoperative obstruction, one rarely finds omentum available, the inflammatory focus having previously attracted it.

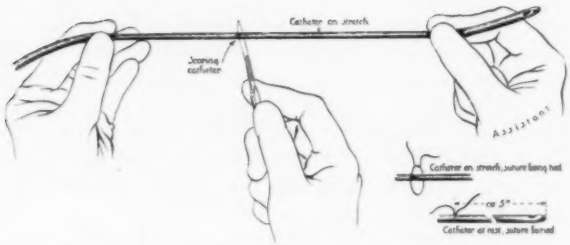

Fig. 10.—Method of scoring catheter to fix it in the bowel.

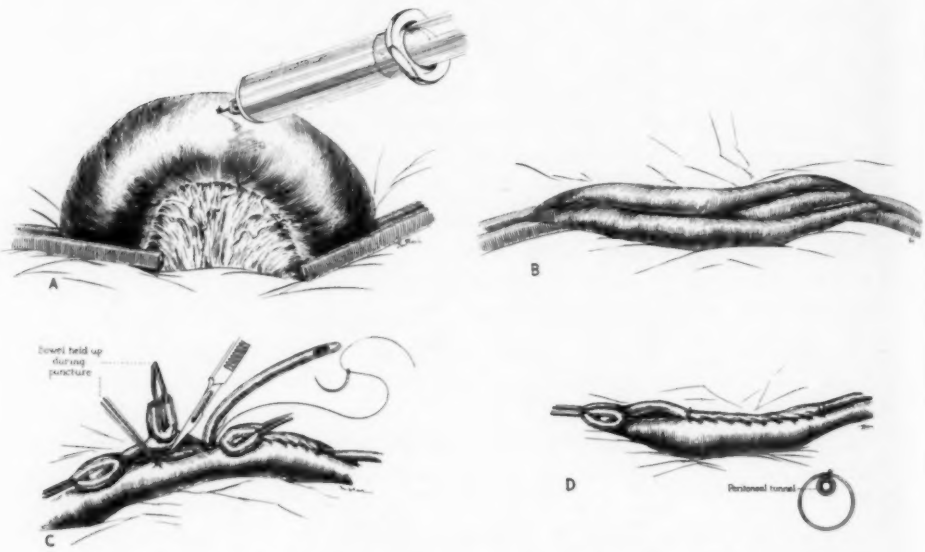

Fig. 11.—Technic of enterostomy: *A* shows the emptying of an isolated loop of its content to permit of an aseptic enterostomy. Usually the greater content can be "milked out" between the fingers. The loop should be completely evacuated with an aspirating needle, however, before any sutures are placed. *B* shows the condition of a much dilated and thinned out intestinal wall immediately after aspiration. *C*, a no. 14 (French) urethral catheter is laid on the bowel and a running stitch of 000 chromic catgut enfolds the bowel about it over a length of 1½ inches; a tiny puncture is made and the catheter introduced into the lumen; the fixation stitch (scoring) is placed and the peritoneal tunnel is continued for another 1½ inches. *D*, the suture is completed; the insert shows the peritoneal tunnel within the bowel in which the catheter lies.

generous lengths of intestine on either side of the devitalized segment are simultaneously removed, the danger of subsequent death from peritonitis is great.

Only under unusual circumstances, such as gangrene of the greater length of the small bowel, is a primary anastomosis to be done. The prohibitive mortality of doing a resection with anastomosis in the presence of devitalized bowel is well illustrated in strangulated external hernias. At the Friedrichshain Hospital in Berlin between the years 1903 and 1922, 1,795 persons with strangulated external hernias were treated, of whom 280, or 15.6 per cent, died. In 1,509 cases without gangrene there were 136 deaths (9 per cent); of the 286 persons

Fig. 12.—Exteriorization of the bowel when nonviable in strangulation obstructions. The gangerenous bowel is cut off after the skin is closed and catheters are tied into each loop. The continuity of the bowel is restored by a secondary anastomotic procedure.

with gangrene necessitating resection 144, or 50.4 per cent, died! Frankau recently reported the results of a collective investigation instigated by the Association of Surgeons of Great Britain and Ireland on strangulated hernia. The mortality for the group was 15.7 per cent. In 105 instances, because of gangrene of the bowel, resection was done, with 45 deaths, a mortality of 42.8 per cent.

The devitalized segment should be brought out through the incision,[13] and on completion of the skin closure, the dead bowel is to be cut off

13. In strangulated external hernia, when the patient's condition permits, the hernia should be repaired after exteriorizing the intestine through a separate short midline incision.

and a catheter tied into each loop (fig. 12). By this means the intestinal content escaping through the proximal catheter may be collected and allowed to run into the distal loop by the gravity drip method. After about six to eight days, it becomes difficult to keep the catheter in the proximal loop satisfactorily, and a secondary anastomosis should be done as soon as the patient's condition will permit. Children particularly do not tolerate well the presence of a high fistula. Its persistence for some time without satisfactory replacement of the escape from the proximal loop into the distal will result in a severe nutritional disturbance that may prove difficult to combat.

SUMMARY AND CONCLUSIONS

Early recognition of acute intestinal obstruction is of paramount importance in treatment. A block in the continuity of the bowel is best identified by establishing the presence of intestinal colic, viz., the audition of loud intestinal noises with the stethoscope at the acme of intermittent crampy pain.

All instances of strangulation obstruction are to be subjected to immediate operation. When nonviable bowel is found, it is far safer to exteriorize the devitalized segment and establish the continuity of the bowel secondarily than to effect a primary anastomosis after resection.

In many patients with acute simple intestinal obstruction decompression can be performed by suction siphonage with a nasal catheter. Instances of adhesive obstruction are particularly amenable to relief by this method. Many patients with late simple obstruction (intrinsic) in which the cause continues to operate can be improved as operative risks by preliminary decompression with the duodenal tube and suction. Inspection of roentgen films of the abdomen is the best criterion in determining whether the decompression is progressing satisfactorily. Of twelve patients with acute simple obstruction observed since the method was first used, successful decompression has been obtained in nine; one of these had a stricture in the terminal ileum, another was a hemophiliac in whom the obstruction was due to hematoma in the bowel. The obstruction in the other seven instances was of adhesive nature, in three cases of remote origin and in four recent. In two of the three instances in which drainage by catheter was not successful enterostomy was done, though in one of these satisfactory decompression was obtained with suction alone. The other patient died following an attempt to relieve the obstructive mechanism and to reestablish the continuity of the bowel. Decompression has also been employed successfully in several instances of subacute and chronic obstructions and as an auxiliary agent after the operative relief of strangulation obstructions. In obstructions of the left half of the colon (usually carcinoma) with

ballooning of the proximal colon with gas, the method is not recommended; in such instances cecostomy or appendicostomy should be done.

The operative treatment of acute intestinal obstruction is briefly discussed, and my method of performing enterostomy is described. Spillage or contamination during the operative manipulations is synonymous with failure!

The value of saline solution and transfusions of blood in the treatment of acute intestinal obstruction is reviewed. Saline solution exhibits the virtues of a specific only in high obstructions. In strangulation obstructions, loss of blood into the infarcted loop may be great, and the transfusion of blood is a great boon in aiding the recovery of patients so afflicted.

MECHANICS OF SCOLIOSIS

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The essential characteristics of the deformity observed in scoliosis have been understood for more than a hundred years. The voluminous literature of scoliosis deals largely with the cause or causes of the deformity, but mainly with the thousand and one methods which have been used in its treatment, with only a few serious studies of the mechanics involved. The striking similarity in the deformities resulting from such a variety of well established causes indicates a uniformity in the intermediate causes, that is, in the mechanics of the deformity. The purpose of this paper is to analyze the previous explanations of this mechanism and to add the results of some original investigations on mechanical models.

ANATOMIC CONSIDERATIONS

The spine is a segmented rod. Each segment consists of a body and a neural arch. The column of bodies is the weight-bearing element, weight bearing including the pressure stresses of all the muscles and ligaments tending to compress the spine. These muscles are attached to the bodies only through the medium of the neural arches.

The axis of all motions between contiguous vertebrae lies in the nucleus of the disk, as may be demonstrated by a careful study of roentgenograms, and as illustrated in Morris' "Human Anatomy."[1] The intervertebral joints are rudimentary diarthrodial, as stated by Smith.[2] Motions allowed are tilting in all planes and rotation. Motion in any one joint is sharply limited by ligaments and articular processes, but the type is double ball and socket, the nucleus pulposus acting as a ball-bearing between adjacent bodies. Use of the term "universal joint" is to be condemned because a universal joint, as the term is used in mechanical engineering, does not allow rotation.

The range of motion allowed in the intervertebral joints is in all directions from the anatomic position of the spine with the normal anteroposterior curves present. The normal anteroposterior curves are therefore no safeguard against lateral deformity.

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1. Jackson, C. M.: Morris' Human Anatomy, ed. 8, Philadelphia, P. Blakiston's Son & Co., 1925, p. 536.

2. Smith, N. R.: The Intervertebral Discs, Brit. J. Surg. **18**:358, 1931.

TYPE OF DEFORMITY

The term "rotary lateral curvature" indicates the two elements of deformity commonly recognized. Lateral curves are labeled right or left to indicate the side of the convexity. In the presence of a primary curve to one side, secondary curves develop which tend to bring the two ends of the spine back into the same planes and preserve general bodily alinement.

Rotation is an integral part of side bending. The bodies rotate toward the convexity of each curve, so that the lateral deviations of the column of bodies are greater than the deviations of the spinous processes. Rotation is greatest at the apex of each curve and tends to be greater in the primary than in compensatory curves. The degree of rotation observed frequently exceeds by far the degree allowed in a normal spine. In the lumbar spine, where normal rotation is almost nil, rotation of 45 degrees frequently occurs, the observations of Calvé [3] to the contrary notwithstanding. As in paralytic clubfoot the tarsal bones grow into a shape allowing more and more deformity, so in the spine the maintenance of a position at one extreme of normal range of motion causes young bone to adapt itself to greater and greater degree of deformity. Each isolated vertebra is stated to rotate about a point in space about 1 inch (2.5 cm.) posterior to the tip of its spinous process. As regards the whole column, however, each vertebra rotates on its adjacent vertebra about a long axis passing through the nuclei of the disks.

A third element of deformity has been observed with sufficient frequency in paralytic and idiopathic cases to warrant consideration. This is kyphosis. In the dorsal region it appears as an increase in the normal kyphosis and in the lumbar as a decrease in the normal lordosis or as actual kyphosis. Like rotation it tends to be greatest in the primary curve and like lateral curvature it tends to be compensated for by the anteroposterior curve above or below. Several authors have pointed out that the apparent kyphosis in a dorsal curve is partly due to prominence of the transverse processes and ribs, but an actual kyphosis of the column of bodies through the primary curve, as shown in lateral roentgenograms, is the rule. Early stages of idiopathic scoliosis frequently show a simple exaggeration of the normal anteroposterior curves, that is, the picture of a relaxed or weak back. But as lateral curvature progresses, a relative kyphosis through the primary curve becomes apparent in the majority of cases. Some tracings of roentgenograms showing this factor are reproduced in figure 1.

3. Calvé, J.: Some Preliminary Observations on Scoliosis, Am. J. Orthop. Surg. **12**:13, 1914.

MECHANICS OF PRODUCTION

A number of explanations of the mechanics of the deformity have been advanced from time to time. Some of them are conflicting. Their multiplicity in itself urges a critical review.

1. *Inequality of Columns.*—Historically after Dods,[4] whose observations were incorrect, comes the explanation of Meyer,[5] followed by Bauer [6] and lately mentioned by MacLennan.[7] Meyer based his theory on two important observations: that strong ligaments existed, anterior and posterior, but not lateral, to the vertebral bodies, and that the column of bodies was compressible while the column of laminae was elastic. MacLennan mentioned unequal growth of the two columns, but the principle is the same. In either case excess length of the column of bodies would cause, first, hyperextension of the spine. In the presence of lateral curvature, from habitual position or any other

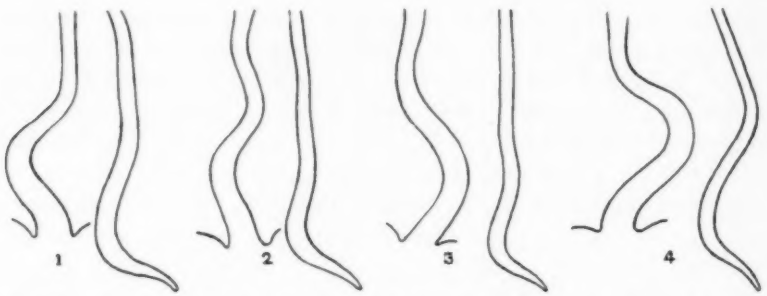

Fig. 1.—Tracings of columns of vertebral bodies from anteroposterior and lateral roentgenograms showing kyphosis, actual or relative, in the primary lateral curve.

cause, the too-long column of bodies would tend to digress from the midline farther than the column of laminae, especially with no lateral ligaments to prevent such deformity.

This ancient theory cannot be wholly denied. Objection to it can be raised on the ground that flexion and not extension is generally recognized as a prescoliotic stage, supported by Lovett's [8] conclusion

4. Dods, A.: Pathological Observations on the Rotated or Contorted Spine Commonly Called Lateral Curvature, London, Cadell, 1824.

5. Meyer, H.: Die Mechanik der Skoliose, Virchows Arch. f. path. Anat. **35**:125, 1866.

6. Bauer, L.: Lectures on Orthopedic Surgery Delivered at the Brooklyn Medical and Surgical Institute, ed. 2, New York, William Wood & Company, 1882.

7. MacLennan, A.: Scoliosis, Brit. M. J. **2**:864, 1922.

8. Lovett, R. W.: The Mechanics of Lateral Curvature of the Spine, Boston M. & S. J. **142**:622 (July 14) 1900.

that rotation first occurs in positions of flexion, and Abbott's [9] system of treatment in flexion. Mechanically, the theory has its undeniable points. It works out on the articulated spinal model. The transference of the anterior longitudinal ligament into a lateral position by rotation might serve to explain the spontaneous arrest of progress of deformity frequently observed. Lateral movement having been arrested in this manner, deformity could still progress in a direction lateral with respect to the rotated vertebrae or lateral and backward in relation to the trunk as a whole, which would be in line with clinical observation. I am inclined to think that this mechanism conspires with two others, one of which is responsible for starting the deformity in the same direction.

2. *Articular Facets.*—Adams [10] and Shaw [11] believed rotation due to changes in the posterior articular processes. Exception was taken to this a few years later by Judson [12] on the ground that rotation occurred early in the deformity while changes in the facets were seen only in late cases, and he concluded that such changes were secondary. It seems highly improbable that the facets could cause rotation in the direction in which it is observed. The lumbar facets allow almost no rotation, certainly favor none. The dorsal interarticular joint spaces form an arc with its center somewhere near the nucleus pulposus. But the surfaces lie in a plane passing upward and forward. If two dorsal vertebrae are taken, the upper tilted to the right on the lower and the interarticular facets kept in contact throughout the maneuver, the inferior facet on the right side of the upper vertebra will move downward and backward while that on the left will move upward and forward, producing rotation toward the right or concave side, a phenomenon opposite to that observed in scoliosis.

It must be concluded that the dorsal facets favor physiologic rotation of the entire trunk, as in turning to look behind, but serve only to limit the type of rotation associated with lateral deformity in scoliosis.

3. *Pressure of Ribs.*—No review of theories of rotation should omit the classic work of Feiss.[13] This author conceived the trunk as a hollow tube and argued as follows: Bending a tube to the side produces tension on the convex and laxity on the concave side. Tension

9. Abbott, E. G.: The Mechanics of a Plaster Corset in Lateral Curvature of the Spine, Am. J. Orthop. Surg. **12**:30, 1914.

10. Adams, W.: Lectures on the Pathology and Treatment of Lateral and Other Forms of Curvature of the Spine, ed. 2, London, J. Churchill & Sons, 1882.

11. Shaw, J., quoted by Adams [10] and Judson.[12]

12. Judson, A. B.: The Causes of Rotation in Lateral Curvature of the Spine, Tr. New York Acad. Med., April 6, 1876.

13. Feiss, H. O.: The Mechanics of Lateral Curvature, Am. J. Orthop. Surg. **4**:37 (July) 1906.

on the convex side pushes the ends of the ribs toward the concavity. The ends of the ribs push the posterior parts of the vertebrae toward the concavity, thereby rotating the anterior part or bodies toward the convexity. Feiss called this a retrograde or reactionary tendency. Some doubt is cast on this theory when it is remembered what a flexible member the rib is in a young child and more when one reflects how difficult it is to reverse the process, to reduce spinal rotation by pressing on the ribs. Lovett [8] held the opposite and more reasonable view when he said: "The ribs, of course, follow the rotation of the vertebrae to which they are attached." The classic case reported by Hoke [14] and recent reports by Sauerbruch [15] and Frey [16] indicate that pressure on the ribs may be used to correct a deformed spine, but it seems improbable that this method represents a reversal of the original mechanism.

4. *Mast and Stays.*—The spine, with its muscular and ligamentous support, has frequently been compared to a mast with its stays. The action of the transverse traction muscles, as described later in this paper, warrants this comparison. The analogy is dangerous only if used to cover all the mechanics of the spine.

5. *Bow and Strings.*—The spine has been compared to a bow. It has been argued that if a strung bow is pushed sidewise at its middle the bow will rotate rather than bend in a second plane. Of course it will, but the entire bow, the middle and both ends, will rotate, a phenomenon which bears no analogy to the scoliotic spine in which the ends remain in their original plane and rotation takes place through a limited area only and in the typical manner described. The spinal mechanism does partake of many qualities of a bow. The flexor muscles represent one long bow-string and the extensor muscles a series of short bow-strings. The significance of this difference will be discussed later. The analogy to bow-strings is satisfactory if used carefully.

6. *Child's Blocks.*—The most simple of all theories is that advanced by Dickson,[17] who said that the rotation reaction is similar to that which occurs if one takes eight or ten child's building blocks stacked one on top of the other in a column and, holding them in the two hands, brings pressure on them in such a manner as to exert more

14. Hoke, M.: A Study of a Case of Lateral Curvature of the Spine, Am. J. Orthop. Surg. **1**:2, 1903.

15. Sauerbruch, F.: The Surgical Treatment of Severe Scoliosis, Arch. f. klin. Chir. **118**:550, 1921.

16. Frey, R. K.: Surgical Treatment of Dorsal Scoliosis, Deutsche Ztschr. f Chir. **169**:13, 1922.

17. Dickson, F. D.: Operative Treatment of Lateral Curvature of the Spine, J. Missouri M. A. **24**:1, 1927.

force on one side than the other. When this is done the column bends and at the same time the individual blocks rotate on each other, this rotation being away from the side of greatest pressure and toward the side of least. This is exactly what vertebrae do when exposed to unequal pressure because of scoliosis. The only difficulty is that blocks do not behave in such a fashion. I have experimented with children's blocks for hours without being able to observe any such rotation. And I can see no reason to expect such a phenomenon.

7. *Flat Flexible Rods.*—The whole problem has been repeatedly dismissed with the magic formula "flexible rods." Both Dick [18] and Guérin [19] formulated a theory which was briefly stated by Bigelow [20] in 1844 as follows: "The principle of torsion rotation is illustrated by bending a blade of grass or a flat flexible stick in the direction of its width. The center rotates on its longitudinal axis to bend flatwise in the direction of its thickness." Smith [21] attacked this theory in 1931, pointing out that Bigelow's statement "implies that (1) there is continuity of substance in the spine, (2) the spine is flat, (3) the plane of flatness is in the frontal plane, (4) rotation is a concomitant element of lateral flexion." Smith attempted, however, to support the analogy of the "blade of grass" by including the idea of tripedal support, stating that "a cross section of the spine is not rectangular but triangular, with its greatest diameter in the antero-posterior plane. The amplitude is less in sagittal bending than in lateral bending. Any tendency to rotation is greater in sagittal than in frontal flexion."

The first two premises outlined by Smith are patently false. There is neither continuity nor flatness in a series of double ball and socket joints. There is an element of truth in the tripedal quality of the spine, whether the posterior points are represented by pressure between articular facets in extension or by tension of the capsules of these joints in flexion. The nucleus and the articular facets do form a triangle. But the side connecting the facets is the shortest side only in the lumbar and lower dorsal regions. In the upper dorsal and cervical regions this is the longest side, as may be seen from inspection of typical vertebrae from these regions (fig. 2). Yet rotation with scoliosis occurs in the same fashion in both regions.

In order to comprehend fully the "blade of grass" idea some experiments were performed with flat flexible rods. A strip of stiff card-

18. Dick, H.: A New Instrument for the Treatment of Spinal Curvature, M. Times & Gaz. **2**:738 (Aug. 20) 1864.

19. Guérin, J.: Mémoire sur l'étiologie générale des déviations latérales à l'épine par rétraction musculaire active, Paris, au bureau de la Gaz. méd., 1840.

20. Bigelow, H.: Orthopedic Surgery, Boylston Prize Dissertation, 1844, Boston, W. D. Ticknor & Co., 1845, p. 168

21. Smith, L. D., in discussion of Carey.[28]

board 17 inches (43.1 cm.) long and five-eighths inch (1.6 cm.) wide was mounted in a vertical position with provision for lowering the upper end to allow bending. Both ends of the flat rod were mounted on universal joints to allow movement in two planes so regulated that the deviation of both ends from the vertical should be equal (fig. 3).

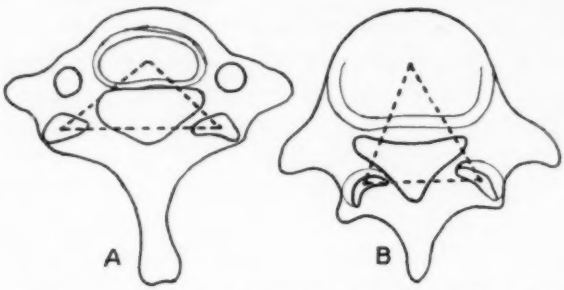

Fig. 2.—*A*, seventh cervical vertebra. *B*, fifth lumbar vertebra.

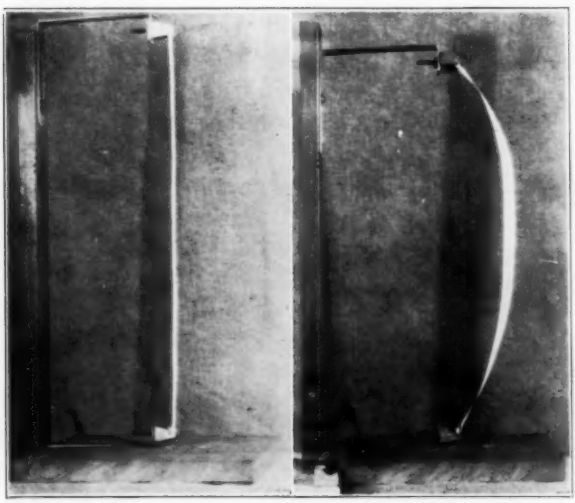

Fig. 3.—Rotation secured in an experiment with a flat flexible rod.

When the upper end was lowered, the cardboard bent in its thickness. When the joints above and below were set at 45 degrees in opposite directions to bend the cardboard in the diameter of its width, rotation took place in such a way that the concave edge bent farther out from the vertical in the plane of its thickness. Interpreted in terms of the spine this phenomenon is as follows: If the width of the cardboard be regarded as in the sagittal plane of the spine, kyphoscoliosis produces rotation of the anterior edge (bodies) to the convexity of the

lateral curve, as seen in clinical scoliosis. In lordoscoliosis the rotation is in the opposite direction. These two results, incidentally, are in accord with Lovett's [22] observations on movement in the normal spine. But to carry the analogy into the upper dorsal and cervical spine where the greatest diameter of the tripod is in the frontal plane, one must regard the width of the cardboard as representing the frontal plane of the spine. Here kyphoscoliosis produces rotation of the bodies toward the concavity of the lateral curve and lordoscoliosis produces rotation in the direction observed in clinical scoliosis. Yet in clinical scoliosis rotation is always that of bodies toward the convexity regardless of the level of the spine involved. The only conclusion possible from all this is that the spine bears no relation to a flat flexible rod, and that the "blade of grass" analogy is a poor one.

8. *Symmetrical Flexible Rods.*—As one of the conclusions from his notable research into the movements of the spine Lovett [8] stated: "The spine behaves in general as any flexible rod would under similar conditions so far as rotation is concerned."

Z. Adams [23] also said: "The column of bodies obeys the laws of flexible rods." The magic formula "laws of flexible rods" transfers the problem into the realm of applied physics, beyond the comprehension of busy surgeons. In my quest for such abstruse knowledge I sought the assistance of Dr. G. B. Pegram, head of the department of physics at Columbia University. Regarding the spine as a series of double ball and socket joints it may correctly be compared only to a symmetrical rod. A series of experiments was therefore inaugurated, using a thick-walled, small-bore, flexible rubber tube, and attempting to duplicate the conditions present in the spine.

The material used was Du Pont nitrometer tubing, an extremely flexible tube with an outside diameter of five-eighths inch and an inside diameter of one-fourth inch (0.6 cm.). In the first experiment a piece of tube 6 inches (15.2 cm.) long was mounted on a short dowel-pin in the center of a base ruled off with parallel lines (fig. 4). A scarf-pin was stuck through the tube near its upper end, horizontally and parallel with the base lines. A cotton thread looped about a common pin driven through the center of a small cork placed in the upper end of the tube provided a means of manipulating the tube without rotary stresses. Rotation of the tube was to be observed by looking down from above, noting the position of the scarf-pin in relation to the parallel base lines. The following results were noted: Pulling the tip

22. Lovett, R. W.: The Mechanism of the Normal Spine and Its Relation to Scoliosis, Boston M. & S. J. **5**:153, 1905.

23. Adams, Z.: The Relation of Bony Anomalies of the Lumbar and Sacral Spine to the Causes and Treatment of Scoliosis, Am. J. Orthop. Surg. **12**:45, 1914.

forward or backward in the plane of the indicator caused no rotation. Pulling the tip to either side, at right angles to the indicator, caused no rotation. Pulling the tip toward any intermediate point caused an apparent deviation from the parallel base lines when viewed from above. But it was also noted that in any such position the indicator was no longer horizontal to the base. This experiment, therefore, might be considered analogous to side bending of a normal spine, as the results agree in every way with Lovett's [22] observations, but not analogous to scoliosis.

It was thought that conditions of the spine could be represented more faithfully by a compound curve, keeping the two ends of the tube

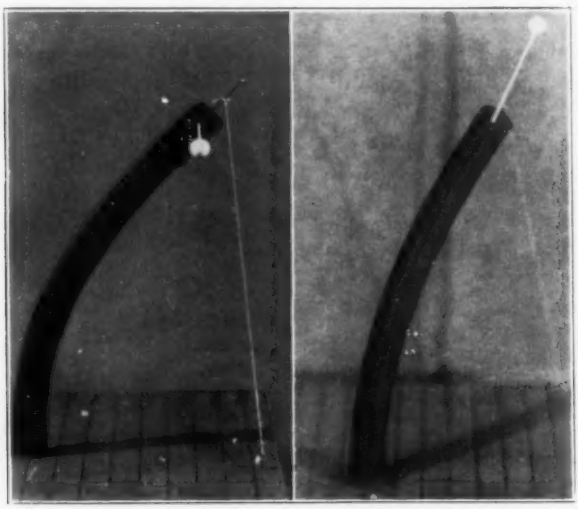

Fig. 4.—Experiment illustrating absence of rotation in a symmetrical flexible rod.

parallel and in the same plane, as they tend to remain in the scoliotic spine. A 17 inch piece of tubing was therefore mounted on a short dowel in the center of a base ruled off with parallel lines, and the upper end on a similar dowel beneath the cross-arm of a standard so arranged that lowering the cross-arm would produce a long curve in the center with compensatory curves near either end (fig. 5). A series of five scarf-pins were stuck through the tube at equidistant points, all parallel with each other and with the parallel lines on the base when viewed from above. The rod being perfectly symmetrical, the bulging of the tube from the vertical, caused by lowering the cross-bar, occurred with equal freedom toward any quadrant of the circle. The primary curve was rotated through the full circle by means of a wire loop passed

loosely about the midpoint of the tube in order to obviate any rotary stress. Looking downward from above, it was observed that throughout the maneuver the pins remained parallel to each other and to the base lines. This result is entirely in accord with the laws of physics. In a symmetrical rod there is no greater resistance to bending in one direction than another, and, therefore, no tendency to rotation.

This, then, disposes of the mystery of "flexible rods" as far as statics of the spine are concerned. A series of ball and socket joints can be compared only to a symmetrical flexible rod. And a symmetrical flexible rod does not rotate at all, either with side bending in extension

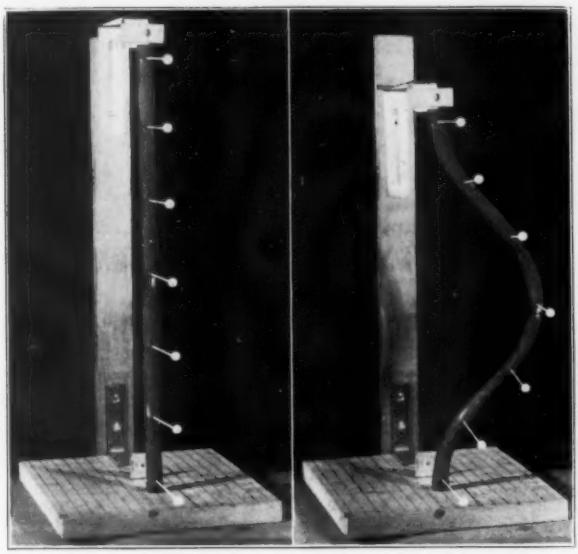

Fig. 5.—Another experiment with a symmetrical flexible rod, showing no rotation in a compound curve.

or with side bending in flexion. In considering the dynamics of the spine the flexible rod will be used again and a different story found.

9. *Muscle Imbalance.—A.* Action of Muscles: So far, I have considered only the static elements of the spine. Muscle is dynamic as compared to bone, cartilage and ligament, which are static. The form of bone, according to the laws of Wolff, which considered gravity only, or according to the remittent back pressure vectors of Carey,[24] which deal with muscle pull, is determined by the stresses placed on it. The static part played by ligaments is demonstrated by the way in which they stretch or shrink, as the need may be, to accommodate themselves

24. Carey, E. J.: Studies in Dynamics of Histogenesis, Radiology **13**:127, 1927.

to altered positions of the spine or of any other joint. Steindler [25] makes the excellent observation that "the ability of the spine to revert at will to perfect anatomic symmetry is the salient characteristic of normal function," and muscle is the only element subject to the will.

Muscles affecting the spine are many and their actions are complex. Several authors have attempted to classify them into more simple groups. Meyer,[26] in 1856, classified them into (1) posterior spinal, (2) anterior spinal (sternocleidomastoid and recti), (3) external oblique and intercostal, (4) internal oblique and intercostal and (5) transversalis. Judson,[12] in 1876, divided them into (1) those nearly parallel to the spine and (2) those connecting the vertebral column with the shoulders, the thoracic and abdominal parietes and the hips. Mackenzie [27] classified postspinal and prespinal muscles, the quadratus lumborum being the only prespinal muscle recognized in the dorsolumbar region. The latest classification, that of Carey,[28] recognizes (1) superficial parallel bow-string muscles, the rectus abdominalis, intercostals and sacrospinalis; (2) deep parallel bow-string muscles, the intertransversarii; (3) superficial transverse traction torsion muscles, the trapezius, latissimus dorsi, serratus anterior and pectoralis major, and (4) deep torsion muscles, the semispinalis, multifidus and long and short rotator muscles.

Of these classifications the old one of Meyer [26] seems the best. Judson's [12] paper dealt primarily with the cause of rotation. He showed a model in which he had mounted an articulated spine inside a rectangular wooden frame. His group of transverse muscles and ligaments were represented by rubber bands connecting the tips of the spinous processes with either side of the frame. A flat flexible brass rod was passed through the spinal canal from the top to the bottom of the frame, its width in the sagittal plane. When this was depressed from the top, it must needs bend to the side. His transverse muscles held the spinous processes in the midline, and the bodies rotated toward the convexity. The same objection may be raised to Carey's [28] recent models, that the spinal muscles are not attached to outside, independent supports but to parts of the trunk which move in coordination with the spine. Mackenzie [27] ignored the most important flexors of the spine, those anterior to the trunk, and classified the quadratus lum-

25. Steindler, A.: Diseases and Deformities of the Spine and Thorax, St. Louis, C. V. Mosby Company, 1929.

26. Meyer, H.: Lehrbuch der physiologische Anatomie, Leipzig, W. Engelmann, 1856.

27. Mackenzie, C.: The Action of Muscles, ed. 2, New York, Paul B. Hoeber, Inc., 1930.

28. Carey, E. J.: Scoliosis: Etiology, Pathogenesis and Prevention of Experimental Lateral Curvature of the Spine, J. A. M. A. **98**:104 (Jan. 9) 1932.

lorum as a flexor. The quadratus is most effective in side bending. But its origin and insertion are both posterior to the nuclei of the lumbar disks, and it can be felt to contract strongly on both sides in pure extension movements. Carey's [28] classification is more anatomic than functional.

I propose classification of the muscles affecting the spine into the following functional groups:

1. Flexors
   - A. Longus colli
   - B. Sternocleidomastoid and other anterior muscles of the neck
   - C. Rectus abdominalis
   - D. Intercostalis externus and obliquus externus
   - E. Intercostalis internus and obliquus internus
   - F. Psoas major

The longus colli has the distinction of being the only muscle, except the diaphragm and a part of the psoas, attached to the spine anterior to the axes of motion, the nuclei pulposi. The important flexors are those stretched over the anterior surface of the trunk, continuous from the skull to the pelvis, with the interposition of sternum and ribs, and not touching the spine at any intermediate point except through the medium of the ribs. These are properly bow-string muscles. The oblique intercostal and abdominal muscles are important rotators of the entire trunk. Their action rotates the spine with the trunk, but does not produce torsion of the type seen in scoliosis. Combined action of the obliques on one side produces pure side bending with no direct rotary effect on the spine. Although the psoas major is primarily a muscle of the lower extremity, it is usually classified as a flexor of the spine and on the articulated spinal model the direction of its pull caused flexion of the lumbar spine in spite of the fact that the bulk of its attachment lies a little behind the nuclei.

2. Extensors
   - A. Interspinales
   - B. Sacrospinalis-iliocostalis lumborum, dorsi and cervicis and longissimus dorsi, cervicis and capitis
   - C. Intertransversarii
   - D. Semispinalis capitis, cervicis and dorsi
   - E. Splenius capitis and cervicis
   - F. Multifidus

As bow-strings the extensors are only a series of short bow-strings. Considered as a group they are attached to each vertebra consecutively. Because of this important contrast to the flexor muscles they had better not be called bow-strings at all. With the exception of the central fibers of the interspinales, all the extensors are attached laterally, as

well as posteriorly to the axis of motion; acting asymmetrically, they produce side bending. The semispinalis cervices and dorsi, both splenii and the multifidus, connecting spinous processes to transverse processes or to the mastoid, also belong to the next group.

3. Deep Torsion Muscles
   A. Rotatores
   B. Multifidus
   C. Semispinalis cervicis and dorsi
   D. Splenius capitis and cervicis

All but the rotatores are also extensors. Their general plan of arrangement is from transverse processes to spinous processes of vertebrae at a variable distance above.

4. Transverse Traction Muscles
   A. Transversus thoracis and abdominalis
   B. Trapezius
   C. Rhomboids
   D. Latissimus dorsi
   E. Sacrospinalis
   F. Quadratus lumborum

This group corresponds to Carey's [28] transverse traction torsion muscles and to Judson's [12] group of those connecting the vertebral column with the shoulders, the thoracic and abdominal parietes and the hips. The muscles connecting the scapula with the shoulder and those connecting the scapula and shoulder with the wall of the chest belong to this group indirectly. Except for the transversi and for the middle portion of the trapezius they are not really transverse but diagonal, and resemble stays supporting a mast.

B. Mechanics of Muscle Pulls: I shall take the last group first, the transverse traction muscles. Judson [12] showed models, as previously described, in which the spine was passively bent to the side and the passive action of transverse bands between the spinous processes and an outside frame caused rotation of the bodies toward the convexity (fig. 6*A*). Carey's [28] models show a spine which tends to remain straight because strung on a vertical spiral spring and which is actively pulled into a curve by the elasticity of springs passing between spinous processes and an outside frame (fig. 6*B*). This kind of pull causes rotation of the bodies toward the concavity.

The important fault in both demonstrations is the hypothesis of independent outside supports as origins of muscles. The arrangement of transverse muscles on a chest supported by a spine is represented schematically in figure 7*A*. In this schema the upper trapezius and levator scapulae and the lower trapezius and latissimus dorsi are represented as symmetrical in order to hold the ends of the curve in the

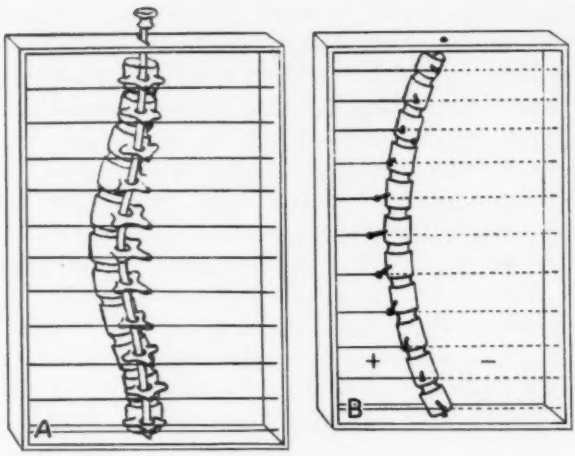

Fig. 6.—*A*, passive transverse pull (Judson). Rotation of bodies toward convexity. *B*, active transverse pull (Carey). Rotation of bodies toward concavity.

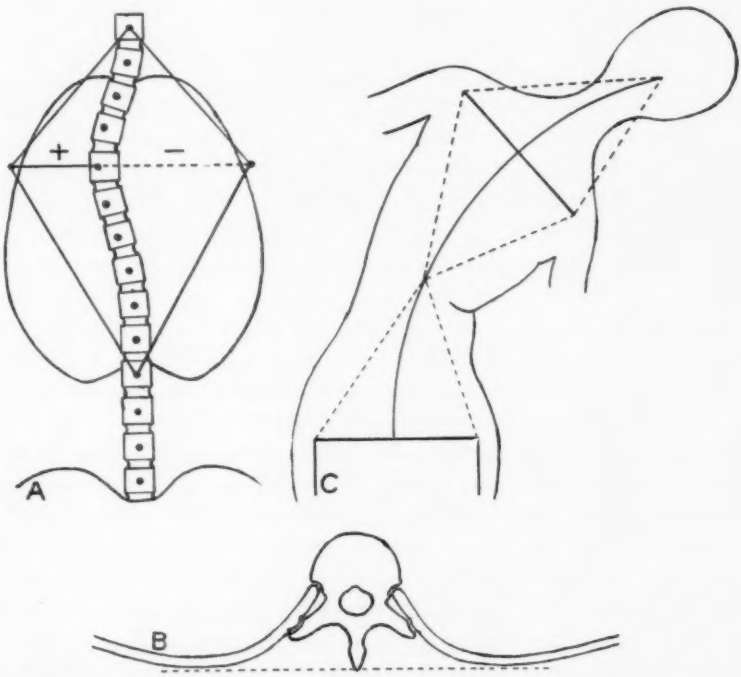

Fig. 7.—*A*, schema illustrating arrangement of transverse muscles on the chest, supported by the spine. *B*, relation of the ribs to the spine and the pull of the transverse muscles. *C*, effect of diagonal muscles.

midline, and the middle portion of the trapezius is stronger on the left than on the right. It is seen that asymmetry of truly transverse muscles produces a curve that is convex on the side of the stronger muscle, with a tendency toward rotation of the bodies toward the concavity. Figure *7B* illustrates how well the ribs, from which the transverse muscles eventually take origin, are locked into the spine by the pull of these very muscles, and at what a marked mechanical disadvantage they are in producing rotation.

The effect of diagonal muscles, the stays of the spinal mast, is represented in figure *7C*. A spinal curve convex to the left is shown, with pelvis and shoulder girdle indicated in their relative positions. For clarity, the three pairs of dotted lines may represent, from above downward, the upper trapezius, the latissimus dorsi and the quadratus lumborum. It is readily seen that all muscles on the left, the convex side, are elongated. The rotation effect of these muscles is dependent on whether they are active or passive. In an upright spine bent in this fashion all the muscles on the left would be active in resisting gravity, and the effect of their pull on the spinous processes would be rotation of the bodies toward the concavity. If gravity were removed, as in side bending from the horizontal position against gravity, the diagonal muscles on the right, concave, side would be actively contracted and effect rotation of bodies toward the convexity. In actual cases of paralytic scoliosis the deformity may be to either side. Steindler,[25] Moffatt [29] and others have noted the capacity of patients for throwing the weight of the trunk toward the paralyzed side, leaving control of movement to sound muscles on the convex side. The rotation effect of this group is slight; I think it better not to call them rotation muscles.

The group classified as deep torsion muscles undoubtedly rotate the spine. The action of each individual muscle is to rotate the entire trunk above, turning the bodies to the side opposite the active muscles. All of them acting on one side would produce a spiral twist of the spine, each vertebra tending to retain its normal relation to the plane of the trunk at its own level. As indicated by Fergusson,[30] for imbalance of these muscles to produce the deformity typical of scoliosis one must suppose weakness of rotators on one side below the apex, and on the opposite side above the apex, of each curve. It is difficult to imagine such a scattered imbalance occurring primarily with the regularity with which the typical deformity occurs. I believe it better to think of this group, aided by the oblique muscles of the chest and

29. Moffatt, B. W.: The Operative Treatment of Scoliosis, J. Bone & Joint Surg. **10**:316, 1928.

30. Fergusson, A. B., in discussion of Carey.[28]

abdomen, as producing spiral rotation of the entire trunk, and to look elsewhere for the cause of that rotation typical of scoliosis.

The flexors and extensors are all arranged more or less parallel to the spine. There is this important difference, that, whereas the flexors are arranged as a bow-string from one end of the spine to the other without contact with intermediate vertebrae, the extensors are attached to successive vertebrae. The mechanical significance of this in reference to rotation can be demonstrated on the flexible tubing previously described as well as on the articulated spine. Figure 8*A* shows the 17 inch tube mounted as before, with flexors represented by a rubber band stretched between the heads of pins placed anteriorly near either end and extensors by short bands connecting a series of pins placed posteriorly. Lowering the cross-bar causes a forward or backward curve, depending on the relative tension of the anterior and posterior bands. Using a loose wire loop to produce lateral curvature from either position produces a rotation of the type typical of scoliosis, that of the bodies toward the convexity. Both anterior and posterior bands tend to remain in a straight line. The anterior band meets no resistance, while the posterior band in its attempt draws the posterior processes toward the midline, rotating the bodies toward the convexity. This effect may be expected from all of the extensors as they are all attached posteriorly to the axes of motion. It accompanies side bending in either flexion or extension, though it is more marked in flexion because the extensors are under greater tension.

The same phenomenon is illustrated in a mount of actual vertebrae (fig. 8*B*). A skeleton of lumbar and dorsal vertebrae is reconstructed by gluing pads cut from a close-meshed rubber bath sponge between the bodies to represent disks. The last lumbar vertebra is mounted on a block of wood with its top surface at 40 degrees to the base. In order to bring this highly flexible column under control, a rubber tube is threaded loosely through the neural canal and attached to a cross-piece above. The spine now tends to assume the normal anteroposterior curves, but is freely movable in all directions within the limits allowed by the articular facets. Flexors are represented by a long rubber band from a nail driven into the anterior surface of the first dorsal to a point in the base corresponding to the symphysis pubis. An elongated toy balloon was interposed between the flexor band and the spine to represent the hollow cavity of the trunk, but was discarded because of the difficulty of keeping it in place during manipulation of the spine. Extensors are represented by short bands joining tacks driven into the tips of the spinous processes. When the spine is pushed into lateral curvature, typical rotation occurs, as with the "flexible rod" model, and for the same reasons.

An even more striking cause of rotation is explained by a study of the mechanics of the extensor muscles. The arrangement is peculiar in that the great mass of muscles lying on either side of the spine produces both extension and side bending, that the extensors are attached

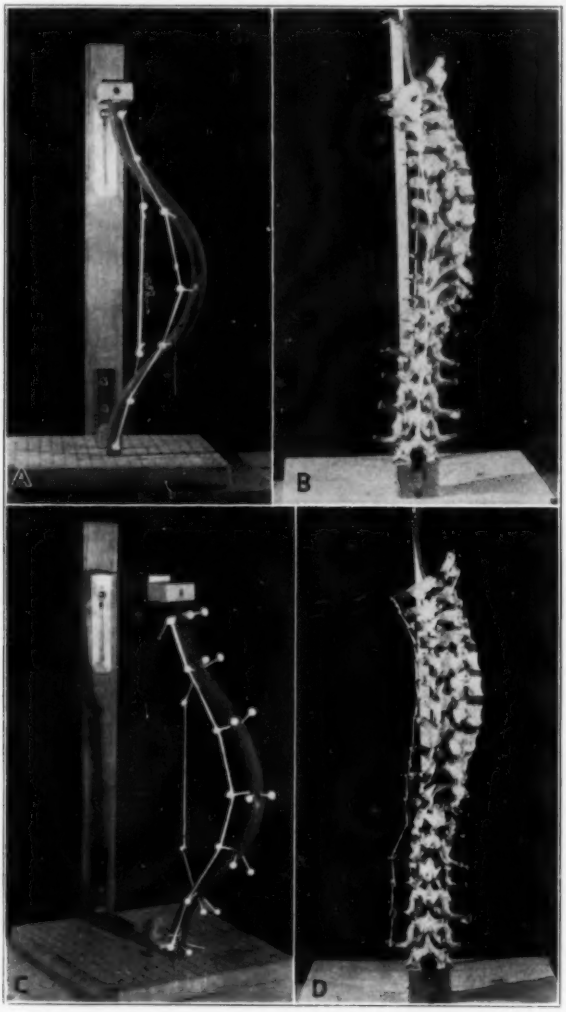

Fig. 8.—*A*, symmetrical flexible rod (rubber tube) mount, illustrating the rotation produced by extensor muscles connected in series as compared with the long bow-string arrangement of flexor muscles, a static effect in lateral curvature from any cause. *B*, same phenomenon produced in a mount of actual vertebrae. *C*, rubber tube mount illustrating the dynamic effect of extensor muscles, relaxation on one side producing lateral curvature, rotation and kyphosis. *D*, same process illustrated in a model of the spine.

lateral to the ball and socket axes of motion and that the side benders are attached posterior to the same axes. The significance of this arrangement may be illustrated on both the symmetrical flexible rod and on the articulated spine. On the rubber tube (fig. 8*C*) a series of common pins is stuck into the tube in the posterolateral plane on either side to represent transverse processes. The band representing flexor muscles is used as before. A series of short bands connecting transverse processes represents the extensors. On lowering the cross-bar, flexion or extension is again determined by the relative tension of the anterior and posterior bands. The rotation effects are more marked in flexion, and as weakness or, better, lengthening of extensors on one side is being investigated, enough tension is used anteriorly to produce flexion. If both extensor bands are put under equal tension no lateral bending occurs. If the right extensor is put under less tension, or removed entirely, lateral bending occurs convex to the right, with extreme rotation of the characteristic type. In removing lateral support from the right, one also removes posterior support. The unimpaired action of the bands on the left not only bends the spine to the right but presses forward the transverse processes on the left. The unimpaired flexors press the unsupported transverse processes on the right side backward. This produces the typical torsion of bodies toward the convexity of the lateral curve.

The process is equally well demonstrated on the spinal model (fig. 8*D*). These last demonstrations fall in line with the observation that typical scoliosis is associated with kyphosis. It is possible on the models to regulate the tension of the flexors and asymmetric extensors in such a way that the extensors on the "sound" side balance the flexors, thus producing lateral curvature and rotation without kyphosis. The body is an amazingly correlated machine which tends to compensate any deformity in every way possible. It seems reasonable, therefore, that this compensatory mechanism may account for the absence of a kyphotic element in some scoliotic spines.

INDICATIONS FOR TREATMENT

The fact that unilateral relaxation of extensor muscles produces the three elements of the deformity, kyphosis, lateral curvature and rotation, is evidence in favor of the treatment of scoliosis by extension. It serves to explain the improvement obtained in the lateral and rotary elements by the prolonged hyperextension secured in recumbency on a convex frame.

Figure 8*D* shows the striking separation of the transverse processes on the convex side which accompanies typical deformity. An operative technic now being worked out has as its object the preven-

tion of all three elements of the deformity by reenforcing the intertransverse ligaments on the convex side.

SUMMARY

1. The joints of the spine are of rudimentary diarthrodial, double ball and socket type, the axes of all motions being located in nuclei of the disks. The normal anteroposterior curve is not a safeguard against lateral deformity.

2. The deformity in scoliosis consists of lateral curvature and rotation. In many paralytic and idiopathic cases a third element, kyphosis, is typical.

3. Theories of the mechanism of the deformity are reviewed. Typical deformity is not due to changes in articular facets or to pressure from ribs. It is not explained by analogies to a mast with its stays, to a bow with its bow-string or to a column of child's building blocks. It is not explained by the physical laws of flexible rods, either flat or symmetrical. One inherent static property of the spine does favor the typical rotation in side bending, i.e., the expansibility of the column of bodies and the elasticity of the column of laminae and processes.

4. The important muscles affecting the spine are classified into four functional groups and their mechanical effects analyzed.

5. Two mechanical principles by which muscle pull produces typical rotation are described. The first depends on the long bow-string effect of the flexors as contrasted with the attachment of the extensors in series to successive vertebrae. The second depends on the triple action of the erector spinae, relaxation on one side producing lateral curvature, rotation and kyphosis.

6. Hyperextension is the position mechanically favorable for correction of scoliosis. Reenforcement of intertransverse ligaments is suggested as a method of preventing deformity.

ELECTROSURGICAL INCISIONS

HISTOLOGIC EFFECTS

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CHICAGO

The purpose of the present work was to study the effects on the tissues of the so-called cutting and coagulating currents, in order to ascertain what specific differences in effects might exist between the two, if any, in the hope of determining the probable damage to the tissues and the rapidity of repair after operations with these currents. The specific effects on several types of tissues were investigated. Sections were made in the skin of the frog, rabbit and dog; various incisions were produced in skeletal and smooth muscle, and the type of tissue lesion was described. Some attempts were made to investigate the histology of blood vessel closure by means of electricity.

The present interest in the employment of the so-called "electric cutting current" devolves on the hope and expectancy of attaining two primary technical advantages:

1. The production of a current which will section vascular tissues and organs difficult of surgical approach, with a concomitant control of capillary hemorrhage.

2. The production of an incision which will approximate a scalpel wound in rapidity and strength of healing reaction without excessive fibrosis.

The first consideration is exemplified in the removal of neoplasms from the brain, and in partial resections of the prostate, liver or spleen, while the second has led to experimentation in intraperitoneal procedures, in which postoperative adhesions are particularly undesirable.

For various reasons, the utilization of electrical machines for surgical purposes, as well as for the treatment of internal diseases, unfortunately attracts a type of practitioner inclined toward extravagant claims. Also, unfortunately, the distinction between cutting operations by electricity and electrocoagulation is not always understood clearly by the operative surgeon who is unfamiliar with the indications for, and the limitations of, cutting and coagulating procedures.

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Aided by a grant from the Council on Physical Therapy of the American Medical Association.

From the Departments of Physiology and Experimental Surgery, Northwestern University Medical School.

The surgical effects of oscillating currents result from the resistance of the tissues to the current, which manifests itself in the local production of heat and also in the local destruction of the tissues of various and peculiar types.

Interest in the therapeutic possibilities of these reactions began in the late eighties, and followed the discovery by d'Arsonval [1] that muscular contractions (the so-called faradic response) cease when the frequency of oscillation of a current applied to the body is raised above 10,000 per second. A current exceeding this number of oscillations per second, based on d'Arsonval's findings, has been termed a high frequency current, in distinction from a low frequency current. The term "high frequency current" in technical electrical usage is based on other criteria. D'Arsonval, in 1891, suggested the feasibility of passing a high frequency current of 3 amperes through the human body, without producing any sensation in the patient except that of heat. In the same year, Nikola Tesla [2] demonstrated the production of heat within the tissues, by the application of high frequency currents. Doyen,[3] impressed by this demonstration, conceived the idea of producing a localized destruction of tissues by the resistance of the body to high frequency currents, and, after eleven years of experimentation, he presented to the French Surgical Congress, in 1907, a technic for destroying carcinoma with electricity. This method is now termed "electrocoagulation." He considered this method of coagulation by ohmic heat a more satisfactory method than any employed up until that time.

It must be remembered that Madame Curie announced the discovery of radium in 1898, and a decade elapsed before noteworthy contributions were made in its therapeutic application. In 1895, Roentgen discovered the rays known by his name, so that research on the therapeutic effects of radium, roentgen rays and medical high frequency began almost concurrently. During the decade before 1908, Nagelschmidt [4] had been engaged in perfecting a high frequency machine for the coagulation of malignant processes, and in 1908 he presented this apparatus at the surgical congress at Budapest. He described the localized heating effects of high frequency currents as "diathermia."

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1. d'Arsonval, A.: Action physiologique des courants alternatifs à grande fréquence, Arch. de physiol. norm. et path. **25**:401, 1893; Traité de physique biologique, Paris, Masson & Cie, 1901.

2. Tesla, N., and Martin, T. C.: Inventions, Researches and Writings of Nikola Tesla, ed. 3, New York, The Electrical Engineer, 1894, p. 394.

3. Doyen, E.: Traitement local des cancers accessibles par l'action de la chaleur au-dessus de 55°, Paris, A. Maloine, 1910; Arch. d'électric. méd. **17**:791, 1909.

4. Nagelschmidt, C. F.: Lehrbuch des Diathermie, Berlin, Julius Springer, 1913; Ueber Diathermie, München. med. Wchnschr. **56**:2575, 1909.

These coagulating machines are not to be confused with those producing cutting currents, which seem to be an American contribution to electrosurgery, the first being demonstrated by Wyeth,[5] of New York, before the surgical section of the New York State Medical Society at Rochester in 1924.

It is my purpose to deal with the distinctions in effect on tissues of the cutting current and the coagulating current, so far as such a distinction can be made, and, in a rudimentary way, with the distinction in the quality of the current which produces the cutting and coagulating effects. The commercial machines produced by various manufacturers for these purposes vary greatly in construction and produce a correspondingly great variety in the qualities of the current elaborated. The ohmic resistance is often too great, or too little, to yield the

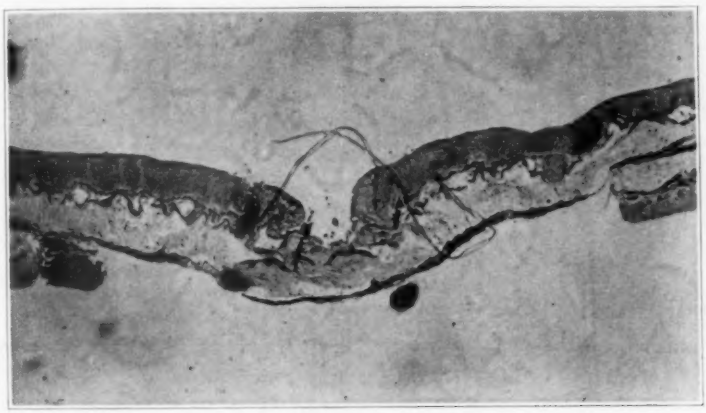

Fig. 1.—Effect of desiccating current on the skin of a frog. The cells near the incision are shrunken without coagulation; × 36.5.

desired effect, and undesired secondary faradic currents, with their resultant neuromuscular response, are induced when improperly constructed machines are employed.

In general, it can be said that raising the amperage produces a coagulating effect, while lowering the amperage and raising the voltage tend to the mechanical disruption of tissue called "electrocutting." The oscillation of current in these surgical machines can be attained in two ways: by means of a set of spark gaps or by radiotrons (radio tubes). The spark-gap machine produces a current from which it is difficult to eliminate damping. This damping is another factor which tends to produce a coagulating current.

5. Wyeth, G. A.: The Endotherm, Am. J. Electroth. & Radiol. **42**:187 (May) 1924.

THE DAMPED AND UNDAMPED HIGH FREQUENCY OSCILLATIONS

In high frequency current, one deals with oscillations of electrons which perform a pendulum-like movement. As the excursion of a pendulum gets slowly smaller and smaller, on account of friction, gravity and air resistance, in spark-gap machines the oscillations of the electron decrease slowly; they are damped in the gap by the air resistance. We deal, accordingly, in spark-gap machines, with damped high frequency oscillations.

Undamped oscillations may be generated by means of radiotron machines. These machines use the same radiotrons which are used for the wireless and for broadcasting. It is known that in radiotrons a pure current of electrons is flowing, which is rhythmically influenced

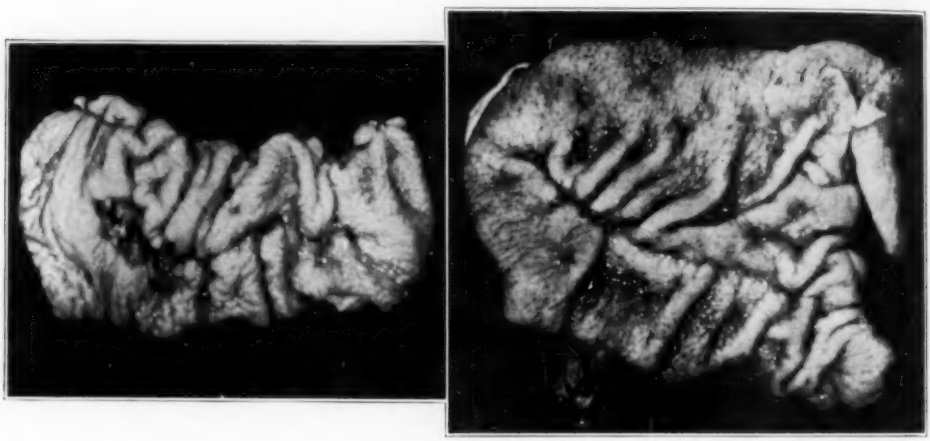

Fig. 2.—The picture on the left represents the average healing of the mucosal surface of the stomach five days after electrical incision; that on the right, a scalpel incision at the same period.

by the continually changing charge of the grid between annode and cathode in the tube. This is the way the oscillations are generated. Since the radiotron is evacuated as far as possible—i. e., free from air—the oscillations are not damped by air or other resistance and are undamped oscillations.

The oscillating currents in radiotron machines have a precisely determined wavelength. In contrast to this, the oscillations of the spark-gap machines are composed of a mixture of waves of different lengths. The present lack of standardization is unavoidable because of the difficulty in measuring accurately the amperage or voltage of a high frequency current during the operation of the machine, so that the proper strength of current used for coagulation, or for cutting, must be learned by experience with each individual machine.

A nice balance must be maintained between voltage and amperage. Also, the capacity inductance and resistance must enter into the determination of the particular current used.

Waller [6] has studied the temperature at which a cutting current operates effectively. An undamped current was investigated. A thermocouple was formed by welding a fine gold wire to a copper needle electrode 0.5 mm. from the tip of the needle, and the ends of the thermocouple were connected to a moving coil galvanometer. Although an alternating potential difference of some thousands of volts is used to form the tiny arc of which the needle is an electrode, while cutting the incision into liver tissue the temperature fluctuation was between 50 and 200 C. This is almost the only attempt to make direct measurements of the physical characteristics of the cutting operation. A wide variation would be expected if various tissues were studied, because of the wide variation of electrical resistance of different tissues.

The production of a cutting current with little or no coagulating effect is now being widely discussed in the German literature by Döderlein,[7] in the Gynecological Clinic of the University of Munich, by Kückens [8] and by Keysser,[9] a former associate of Lexer. The principal French contributors are Champy and Heitz-Boyer.[10]

GENERAL CHARACTERISTICS OF THE CUTTING CURRENT USED ON TISSUES

The technic of electrical cutting is the most recent development in electrical surgery. The electrode glides through the tissues without the necessity of pressure. One has the impression that the tissue melts under the influence of the electrical current. Kirschner,[11] for this reason, has given it the name of "melting cut." This incision has the

6. Waller, M. D.: The Temperature of the Endothermic Knife, Brit. J. Radiol. **4**:178 (April) 1931.

7. Döderlein, G.: Das Schneiden mit den elektrischen Funken, Deutsche med. Wchnschr. **52**:59 (Jan. 8) 1926.

8. Kückens, H.: Ueber einige bei den Diathermieoperation auftretende Gewebeveränderungen und über ihre Bedeutung, Beitr. z. path. Anat. u. z. allg. Path. **85**:695 (Nov. 20) 1930.

9. Keysser, F.: Die Elektrochirurgie, Leipzig, Gustav Fischer, 1931.

10. Champy, C., and Heitz-Boyer, M.: Mécanisme d'action du bistouri électrique à haute fréquence (effets thermiques et mécaniques des courants de haute fréquence sur les tissus), Compt. rend. Acad. d. sc. **189**:1039 (Dec. 2) 1929; Étude des effets mécaniques des courants de haute fréquence: Leur action hémostatique sur les vaisseaux, ibid. **189**:1328 (Dec. 30) 1929; Sur l'action hémostatique du bistouri électrique, Compt. rend. Soc. de biol. **103**:385 (Feb. 14) 1930.

11. Kirschner, M.: Aus der Praxis des "elektrischen Operierens," Klin. Wchnschr. **9**:725 (April 19) 1930.

gross appearance of a scalpel cut. Kelly [12] has called this procedure "acusection," and Keysser [9] has described it as "aukutomy." The histologic effects on tissues can be described in zones, the innermost being a zone of mechanical disruption of tissue and explosion of cells. The mechanics of this disruption has not been determined. The question of whether this effect is produced by molecular dissonation, due to the assumption by the molecules or atoms of the tissues of a new rate of vibration, causing dissolution of the molecular structure, as postulated by Oudin,[13] or is merely a thermal effect due to the sudden expansion of the cell when its liquid contents are converted into steam, which Jellinek [14] thinks the simplest and most logical explanation, is speculative and need not be discussed. Just outside of this zone of tissue disappearance is a zone of elongation or attenuation of cells, this drawing-out effect being seen principally in the nuclei, which are more fluid than the cytoplasm. Kawamura [15] described radiating lines of similarly attenuated cells running out from areas of electrical injury in fatal cases. Depending on the fluidity of the tissues, there is a varying amount of change in the orientation of these elongated cells, so that they tend to come to lie parallel with the direction of the cut. This effect is seen at its maximum in soft connective tissue or muscle, and cannot be produced in the stratum corneum of the skin. In parenchymatous organs, this elongation is transmitted along the nearby blood vessels, perhaps because the tissue surrounding the vessels is less resistant than the vessel wall itself.

Wildermuth,[16] assuming the resistance of a chemically pure physiologic solution of sodium chloride of a temperature of 18 C. as 1, estimated the specific resistance of the various tissues as follows:

Fatty tissue	..	19.4
Brain tissue	5.5—	6.8
Pulmonary tissue	3.5—	4.0
Liver tissue	2.8—	3.3
Skin	2.5—	3.0
Muscle	1.2—	1.5
Blood (approximate)	..	1.0

The higher the fluid or blood content of an organ and the lower its fat content, the less is its resistance to electric section. The great

12. Kelly, H. A., and Ward, G. E.: Radical Breast Operation with Endotherm Knife, Ann. Surg. **42**:88, 1926.

13. Oudin, cited by Lowry, F. P.: The Oudin Current, Am. J. Phys. Therapy **7**:489 (March) 1931.

14. Jellinek, S.: Biologische Wirkungen ultrakurzer Wellen, Wien. klin. Wchnschr. **43**:1594 (Dec. 25) 1930.

15. Kawamura, I.: Elektropathologische Histologie, Virchows Arch. f. path. Anat. **231**:570, 1921.

16. Cited by Keysser.[9]

resistance in cutting through fat, as compared to muscle and skin, at first disturbs the surgeon habituated to the scalpel, which cuts fat more readily than muscle and skin. The variation of resistance to electric cutting necessitates a readjustment of the machine as one passes from skin into fat, and then into muscle or parenchymatous organ.

The Dehydrating Cut.—By a slight change in voltage and amperage, a cutting current can be produced with enough coagulating characteristics to seal the smaller blood vessels along the edge of the cut in an area of so-called "dehydration." This zone is white and presents cells

Fig. 3.—Incision through the wall of the stomach with a cutting current, without a zone of coagulation; × 15.5.

with shrunken contour and pyknotic nuclei, the fluid contents having evaporated. The skeleton contour of the cells is distinguishable. The cytoplasm stains vividly, while the nuclei are hyperchromatic.

GENERAL CHARACTERISTICS OF THE COAGULATING CURRENT USED ON THE TISSUES

This tissue effect is produced *par excellence* by a high amperage and a strongly damped current in a spark-gap machine, or by raising the voltage and amperage of the radiotron machine. A lower frequency of oscillations is necessary than for cutting purposes. Widespread

coagulation can readily be produced with a spark-gap machine without charring or carbonification of the edges of the wound. In the use of this, as in the cutting current, the electrode is brought in contact with the tissues, and then the circuit is closed. If the circuit is closed before the contact is made, sparking across from the electrode to the tissues may result in charring. This zone of charring interferes with the dissemination of the coagulating current and limits the coagulating effect. It is also improper to remove the electrode from the tissues until the current is opened. This sparking from the electrode to the tissues, besides the charring effect, also induces faradic extra currents and muscle jerking which interfere with the delicacy of the operation. Small areas of coagulation assume the form of a half globe or an inverted cone, the base of which is on the surface of the tissue being cut. Two definite zones can be described in the coagulating effect. The inner zone is blanched, and the zone external to this is hyperemic in appearance. The cytoplasm of the inner zone, in histologic preparations, presents a blue nuclear staining, e. g., hematoxylin, contrasting with the red eosin staining of the intact tissue. The tissues so affected are somewhat shrunken, and the nuclei have lost their definition and stain poorly, or disappear. This is the typical picture of coagulation necrosis. The proteins are split, freeing blue-staining acid radicals with a probable increase in the local hydrogen potential. According to Döderlein, the protein hydrosol is changed into a hydrogel. This inner zone passes over into an ill defined outer zone of shriveled cells with pyknotic nuclei and a region of dilated vessels. "The faster the tissue dies, the more it conserves its original form and shape" (Ernest [16]). The primary effect of the heat is identical with necrosis of ordinary skin burns. This applies to preparations made immediately after the operation. Preparations made several hours or days later show the outer zone being invaded by fibroblasts, with round cell accumulation and foreign body giant cell formations. Robertson and Boyd [17] have isolated from the tissues coagulated by heat two immunologically specific toxic proteins. One, diffusible and thermostabile, they term a "neurotoxin," and the other, thermolabile and colloidal, a "necrotoxin." Pfeiffer [18] and Davidson and Mathew [19] have isolated a toxic protein from the urine. It is probable that these toxins produce the outer area of secondary necrosis.

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17. Robertson, B., and Boyd, G.: Toxemia of Severe Superficial Burns in Children, Am. J. Dis. Child. **25**:163 (Feb.) 1923.

18. Pfeiffer, H.: Ueber die Ausscheidung eines peptolytischen Fermentes im Harn bei verschiedenen Formen der Eiweisszerfallstoxikosen, München. med. Wchnschr. **61**:1329 (June 16) 1914.

19. Davidson, E. C., and Mathew, C. W.: Plasma Proteins in Cutaneous Burns, Arch. Surg. **15**:265 (Aug.) 1927.

Nieden [20] has pointed out that deep coagulation, even in the inner zone of complete necrosis, is always uneven. In experiments in association with the physicist Weiss, he tried to account for the unevenness and the direction of penetration according to the distribution of stream lines produced by different shapes of electrodes and marked variation of resistance displayed by the various tissues. This variation makes the direction of penetration and deep coagulation unpredictable, and the resulting injury to the tissue perhaps the most dangerous factor one deals with in electrosurgery. After coagulation in the vicinity of large vessels, a fatal postoperative hemorrhage may occur as the result of an unexpected necrosis of the vessel wall.

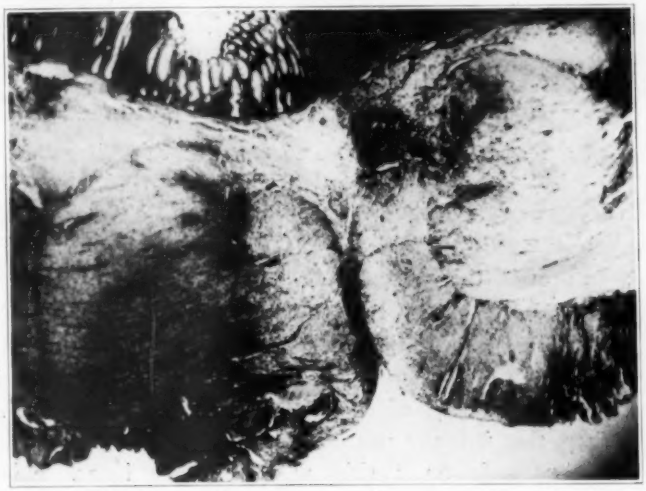

Fig. 4.—Healing of the wall of the stomach after cutting a current incision without a zone of necrosis; × 16.

SPECIFIC EFFECTS ON DIFFERENT TISSUES

Skin preparations were first studied to determine the type of injury produced by the minimum amount of cutting and coagulating current which would leave a visible impression on the surface. Later, attempts were made to incise the skin of the human being, dog, rabbit and frog with a current producing a purely cutting effect without necrosis. In all skin possessed of a stratum corneum, i. e., in all types employed except that of the frog, a specific cutting effect without coagulation could not be accomplished. A narrow margin of coagulation bounded every incision, although this was narrower (a minimum of 1 mm.) with the cutting than with the coagulating current. The stratum

20. Nieden, A.: Das Anwendungsgebiet der Elektrochirurgie, Zentralbl. f. Chir. **30**:1897 (July 27) 1929.

corneum, then, resists the disruptive effect of the cutting current until enough heat is produced by tissue resistance to cause coagulation. At the time of incision, the epidermis is blanched and is thinner because it is shrunken. The individual epithelial cells can no longer be discerned plainly in the stratum corneum, while in the strata mucosa and germinativa the cells assume somewhat the appearance of those in a normal corneous layer. The cells in the basal layers are shrunken together and have dark, spindly nuclei. The fibers of the connective tissues of the corium are either coagulated adjacent to the cut or have lost their fibrillary structure and appear as conglomerate masses, often resembling hyalin in appearance. They can no longer be dyed red in a hematoxylin-eosin preparation, but stain slightly blue. Between these close-pressed masses appear shrunken nuclei. When the cut reaches into the fat, only the connective tissue of the fat is changed, the fibers being broadened and dyed blue, containing pyknotic nuclei. In the stratum reticulare of the corium, flattened gas bubbles appear. Keysser,[9] and Schridde [21] seem to be the first to describe this phenomenon, and Schridde saw it also in the epidermis. In the skin of the rabbit, connective tissue changes extend around the sweat glands at some distance from the region of the incision, and the hairs are fragmented in the changed area around the cut.

The necrosis produced by the minimum of coagulating and cutting current, which will leave a visible impression on the surface of all the skins provided with a stratum corneum, presents no difference in type of tissue injury from that of coagulation previously described. It is concluded, then, that it is not possible to produce a purely cutting effect without some necrosis on a skin with a horny layer. This slight necrosis, produced by the cutting current, does not, however, always preclude healing by primary intention, as I found in another experiment,[22] performed to test the tensile strength of wound healing on dogs' skins, that 60 per cent of electrically produced wounds showed primary union in comparison with 97.5 per cent of primary union in scalpel wounds. When union did occur, however, the electrically produced wounds were somewhat weaker than those produced by the knife until approximately the twenty-fourth day of healing.

In the midperiod of healing, the electrically produced wounds are notably weaker. At twenty-four days, the two curves have not yet

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21. Schridde, H.: Die elektrischen Strommarken der Haut, Centralbl. f. allg. Path. u. path. Anat. **32**:369 (March 15) 1922.

22. Ellis, J. D.: The Rate of Healing of Electrosurgical Wounds as Expressed by Tensile Strength, J. A. M. A. **96**:16 (Jan. 3) 1931; Attempts to Express Mathematically the Healing of Electro-Surgical Wounds, Surg., Gynec. & Obst. **52**:516 (Feb.) 1931.

approximated, when heavy dehydration is employed. That is to say, the attainment of a maximum of strength is delayed in the electrically produced wounds.

The effects of the cutting current on skeletal muscles and the muscular wall of the stomach and intestines were studied. Incisions may be freely made without any zone of coagulation whatever. There are a change of orientation and shape of the superficial cells and a sealing of the capillaries and lymphatic channels. These incisions heal with approximately the same tensile strength as cuts made with a scalpel and without secondary necrosis or infection. Tests on muscle section and healing show no important weakness of the electrically produced wounds at any period of healing. A comparison in healing of electric and scalpel gastrotomy presents curves of healing for the electrically produced wounds, considered as to both rapidity and time of attainment

Fig. 5.—Electric ligation of the vein, with some shattering and explosive effect, due to sudden application with too great current density; × 15.5.

of maximum strength, entirely comparable to the scalpel wounds of the muscle wall of the stomach.

The explanation of the failure of the skin with a corneous layer to give the uniform primary healing which muscle displays after section with a cutting current and to present a narrow border of necrosis when enough current density is displayed to section such skin, probably lies in the fact that keratin, the principal constituent of the corneum, has a high electrical resistance. It can, in fact, be employed as an insulator. When enough current density is employed to penetrate this layer, some coagulation of the contiguous, less resistant skin layers invariably results.

Primary mesodermal healing presupposes two factors, that is, a deposition of a complete fibrillary fibrin network across the wound and satisfactory cellular fibroplasia. Hartwell [23] and others have shown that

23. Hartwell, S. W.: Surgical Wounds in Human Beings, Arch. Surg. **19**:835 (Nov.) 1929.

fibrin and collagen are deposited in primary healing by a chemical reaction from "prefibrin" by the absorption of the edema fluid of a wound. This appears to them to be entirely or largely independent of cellular activity and conditioned by the hydrogen potential of the wound. Fibroplasia occurs first by stereotropism of wandering tissue cells or clasmatocytes, along the fibrin network, and, later, by proliferation of these cells. This mechanism was described in 1914 by Harrison,[24] and his work has been studied and corroborated recently by Baitsell,[25] Wereschinski[26] and Carrel.[27] The latter has shown that the formative stimulus to fibroplasia is not found in the blood plasma, but is a local product of the injured tissues. Baker[28] and Hammett,[29] working in Carrel's laboratory, found that the formative stimulus does not depend on liberated amino-acids from the wound or adjacent tissues, but is a protein cleavage product containing a sulphydryl group. The elaboration of this essential stimulus for cell multiplication is apparently not interfered with by the cutting current applied to the muscle or stomach, and, in some instances, to skin. The production of a fibrillary fibrin network is generally not demonstrable after electrosurgery. Following the advice of Hertzler,[30] I attempted to demonstrate this network with a Weigert-Pal stain, and later with the Kilschitzski modification, but found only a granular fibrin deposition, such as one sees in wounds healing by granulation.

The coagulating current, applied to skeletal muscle and stomach, produces the same inverted zone of coagulation as described in the deeper layers of the skin. The appearance of the coagulated muscle, after a few days, resembles hyalin in a necrotic area ultimately surrounded by a connective tissue capsule or absorbed and replaced by fibrosis.

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24. Harrison, R. G.: Reactions of Embryonic Cells to Solid Structures, J. Exper. Zool. **17**:521, 1914.

25. Baitsell, A. G.: Origin and Structure of Fibrous Tissue Found in Wound Healing, J. Exper. Med. **23**:739 (June) 1916.

26. Wereschinski, A.: Beiträge zur Morphologie und Histogenese der intraperitonealen Verwachsungen, Leipzig, F. C. W. Vogel, 1925.

27. Carrel, A.: Process of Wound Healing, Proc. Inst. Med., Chicago **8**:62, 1930.

28. Baker, L. E.: Chemical Nature of Substances Required on Cell Multiplication, J. Exper. Med. **48**:163 (Feb.) 1929.

29. Hammett, F. S.: Chemical Stimulus Essential for Growth by Increase in Cell Number, Proc. Am. Philos. Soc. **68**:151 (April) 1929.

30. Hertzler, A. E.: Newer Conception of Wound Healing as Applied to Practical Surgery, Am. J. Surg. **7**:293 (Sept.) 1929; personal communication to the author.

PARTICULAR EFFECTS ON BLOOD VESSELS

The discussion of the effects of these currents on the blood vascular system can be divided, for clinical purposes, into:

1. Capillary hemostasis in vessels of the size which do not usually bleed after the hemostat is removed in ordinary surgical procedure, e. g., laparotomy wounds.

2. The coagulation of moderate-sized vessels, such as the ones which must be ligated before the hemostat is removed.

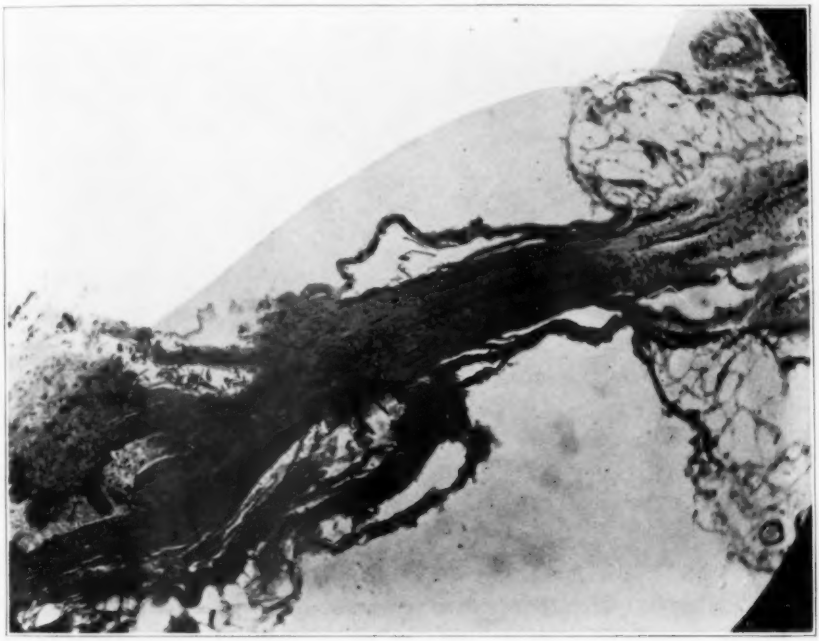

Fig. 6.—Successful hemostasis with uniform contraction of the wall of the artery without necrosis or pigmentation; × 30.

3. The closure of arteries and veins the size of the radial artery or larger.

Hemostasis in Capillaries, Arterioles and Venules.—When tissues are severed with the cutting current without any zone of coagulation, capillary hemorrhage results, which suddenly ceases a few minutes later, even in such vascular tissue as the muscles or the kidney, with a facility that surprises the surgeon. This unusual effect was first investigated by Heitz-Boyer.[31] He noticed, in histologic preparations, the trans-

31. Heitz-Boyer, M.: Action hémostatique, "secondaire" du bistouri à haute fréquence, Bull. et mém. Soc. nat. de chir. **55**:1946, 1929.

mission of elongation and stretching of the cells of the perivascular sheath to a point several millimeters from the cut end of the capillary. There is an abrupt change in the muscular wall of venules and arterioles, causing contraction and fusion with obliteration of the lumen at the point of severance. The arteriole, in particular, often becomes tortuous for a few millimeters. The vascular lumen is occluded by the collapsed walls and the few epithelial lining cells which are torn off and seen free in the lumen. In some specimens, no endothelial changes are seen except a curious, wavelike wrinkling in transverse ridges just at the point where the lumen begins to contract. It did not appear to Heitz-

Fig. 7.—Correct "electrical ligation" of the artery, showing abrupt diminution of caliber. The changes are most notable in the muscular layers; the intima is corrugated but without avulsion of the cells; × 45.

Boyer that the existence of a few avulsed cells in the lumen was sufficient to account for the sudden hemostasis in the capillaries and arterioles after cutting, and he suspected that there must be a rapid and abundant liberation of thrombokinase from attrition of the vascular wall. The following experiment was undertaken. The serum of rabbits' blood was placed in paraffined tubes. Small, well polished pipets were introduced into rabbits' veins and arteries without touching the edges of the wounds of entrance. These vessels were then severed from 1 to 2 cm. from the point of the pipet with undamped current and with the scalpel. The pipets were withdrawn and placed in the tubes containing

rabbits' serum. The pipets from the vessels electrically damaged caused immediate coagulation of the serum, while those from the vessels with the knife wounds caused no coagulation of serum for from ten to twelve minutes, which is the same time that coagulation occurred in the control rabbit serum. He inferred from this that abundant thrombokinase was liberated from the walls of the smaller vessels by the action of the electric current.

Hemostasis in Small Vessels.—Vessels of a size which must be ligated to control hemorrhage in ordinary surgical procedures are not generally successfully obliterated with a cutting current. Either they must be touched with a coagulating current, or the hemostat which

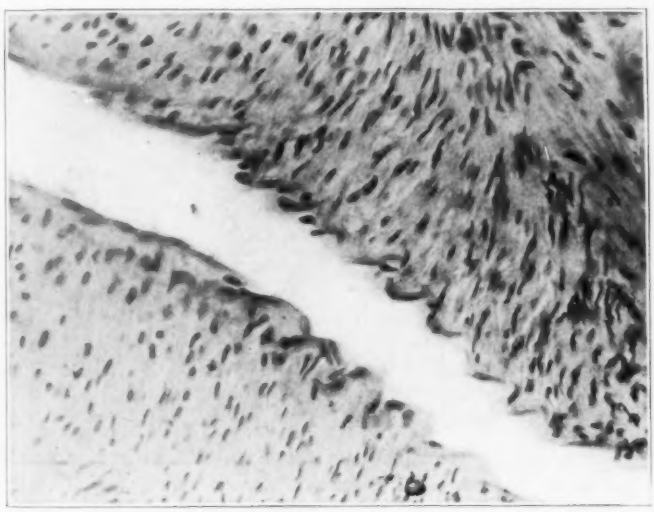

Fig. 8.—Higher magnification to show the typical corrugation without avulsion of the intimal cells at the point of contraction of the vessel; × 65.

occludes them must be touched, in order to produce successful hemostasis. The less tissue surrounding the vessel that is gripped with the hemostat, the less is the area of necrosis formed. The smaller the amount of necrotic tissue, the greater is the chance of absorption of this tissue without a slough, and the chance of infection with secondary hemorrhage from the coagulated vessel. I experimented on coagulation of the splenic vessels and the radicals of the superior mesenteric vessels adjacent to the small intestine in the dog and found that these were best occluded by use of the coagulating current. The walls present the same changes as occur in the capillaries on section. It is possible, however, to exhibit enough coagulating current to break the vessel into fragments and produce hemorrhage, or to explode the brittle, collapsed

and constricted wall a few millimeters from the region of hemostasis. As one becomes more efficient, there is a temptation to employ more voltage for a shorter time. This is dangerous. It is easy to generate a cloud of steam at the point of application, which pushes into the lumen of the vessel and explodes, instead of seals it. It is my opinion that successful coagulation depends on the mechanical effect of constriction of the lumen, and not on thrombus formation. The cells of the muscular layers appear crowded together and stained blue at the point of closure. There are the usual nuclear changes which accompany electric coagulation. The intima is seldom avulsed, and a few free epithelial cells appear in the lumen. If the vessel is coagulated in the solid tissue of an organ, the vessel change does not extend beyond the tissue necrosis in the parenchyma of the organ. This limitation of change was first described by Kuntzen and Vogel.[32] These authors

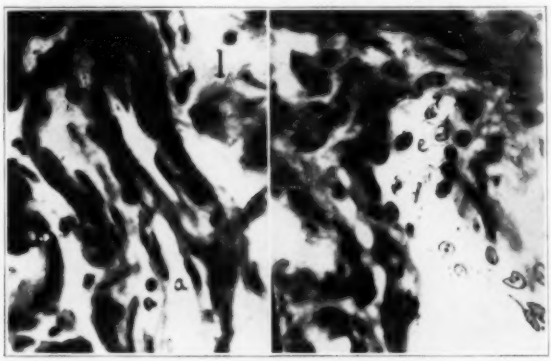

Fig. 9.—*I*, avulsion and elongation of the endothelial cells of a capillary sectioned by a cutting current. *II* represents the more violent action of the coagulating current which sectioned the tissue adjacent to the field shown here. This field is adjacent to the zone of necrosis; × 220.

carried out experiments on rabbits. A lobe of the liver was fixed in the abdominal wall and excised with a coagulating current. The animals were killed on various days. India ink was injected into the portal vein. Thrombi were found to form only as far as the tissue necrosis in the surrounding liver substance. A selective effect of the current on the blood vessels did not manifest itself beyond the region of coagulated liver. No secondary hemorrhages occurred along the vessels. This result agrees with my experience.

Sealing of Vessels up to Six Millimeters in Diameter.—Tinker [33] has described the coagulation of blood in vessels of approximately

32. Kuntzen, H., and Vogel, W.: Histologische Untersuchungen über die Wirkung chirurgischer Diathermiestroeme, Arch. f. klin. Chir. **164**::39, 1931.

33. Tinker, M. B.: Electro-Surgery with Special Reference to Goiter and Malignancy, Ann. Surg. **94**:587 (Oct.) 1931.

6 mm., extending 12.5 mm. along the lumen. I was unable to demonstrate any such thrombosis in the arteries. In veins a definite coagulation extended not farther back than a distance equal to the diameter of the lumen when the vessel was coagulated with a current strong enough firmly to close but not entirely to destroy the vessel. Even after several days this small, red thrombus was still seen in the vessel merely as a coagulation thrombosis, the definition of many of the red cells being retained, and the white cells appeared unchanged in morphology and distribution. There is no progression in the size of the thrombus, when examined several days after the occlusion of the vessel.

CONCLUSIONS

Two distinct types of tissue effect can be produced by electrosurgical machines. These types of tissue effect depend on the qualities of the current employed. The more the damping and the higher the amperage, the greater is the amount of coagulation accompanying the tissue section. Both the cutting and the coagulating current have special indications and advantages in surgical practice. Massive coagulation of tissues with subsequent sloughing of the necrotic area is undesirable as a surgical procedure. The present electrosurgical technic consists of excising a portion of tissue with the cutting or coagulating current.

Two types of electrosurgical machines are in common use; in one the frequency of oscillation is produced by a multiple spark gap and in the other by radiotrons (radio tubes). The first always produces a somewhat damped current. As the damping is diminished the incision more nearly approximates the scalpel cut. As the damping increases the amount of coagulation increases. The radiotron machine can produce a cut without coagulation, as the current is not damped. By increasing the voltage and amperage in this machine, coagulation results. This is likely to be associated with a charring if the amperage is too high. This prevents penetration of the coagulating effect into the tissues.

Experimental cuts were made with both types of current on the skin of the human being, dog, rabbit and frog. The first three of these have a skin with a stratum corneum, and, in the presence of this layer, some coagulation results before penetration of the skin can be accomplished, even with a pure cutting current. This coagulation does not necessarily interfere with primary healing, but only 60 per cent of the cuts produced in dog's skin healed by primary intention. The skeletal muscle and muscular layers of the stomach and intestine can be severed by a cutting current without coagulation. The phenomenon of vessel closure with a coagulating current is interesting, involving collapse,

shrinkage and agglutination of the vessel walls without extensive thrombus formation. A large amount of thrombokinase seems to be liberated in the cutting of capillaries and small vessels. There are many serious errors to be avoided in the coagulating of vessels. Too rapid coagulation causes explosion and subsequent hemorrhage.

In coagulation of tissues, the specific resistance of the tissues varies tremendously; e. g., fat is more than eight times as resistant as muscle, making the direction of the penetration of deep coagulation uncertain and dangerous.

122 South Michigan Avenue.

INFECTION INVOLVING THE ETHMOID, MAXILLARY AND SPHENOID SINUSES AND THE ORBIT DUE TO ASPERGILLUS FUMIGATUS

REPORT OF A CASE

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Infection of the nose and accessory nasal sinuses by Aspergillus fumigatus is a rare disease. In a careful survey of the literature I was able to find only three cases recorded in which the infection in the sinuses was definitely proved to be due to Aspergillus fumigatus and five cases of aspergillosis of the maxillary sinus of undetermined type.

Aspergillus fumigatus was first described by Fresenius in 1775. This is the commonest Aspergillus and is often found in various cereals, straw, hay and similar substances. It is the species most frequently found in man, giving rise to an aspergillosis of various organs. The spores are very resistant. Aspergilli are generally saprophytes, but they may become parasites. The effects on the human organism are due in addition to mechanical irritation to toxins secreted by the fungi. These toxins have been found to act on the muscular and nervous system of dogs. Harmer and Jockes [1] showed that injection of cultures of Aspergillus fumigatus from the antrum into the ear vein of a rabbit caused the death of the animal in seven days. Postmortem examination showed practically all the organs to be affected, and subcultures yielded pure growth of Aspergillus fumigatus. Bodin and Lenormond,[2] in their researches on the poisonous products of Aspergillus fumigatus, working with guinea-pigs, rabbits and pigeons, showed that there are two distinct toxic substances; one produces tetany and convulsions, the other possesses a depressing or paralyzing power.

There are three varieties of Aspergillus which create most devastation—niger, flavus and fumigatus. More infections arise from the fumigatus than from the niger or flavus types. In man, the Aspergillus fumigatus is most commonly found in the ear (external auditory meatus) and in old abscess cavities in the lung. The mode of infection is not definitely known.

From the Johns Hopkins Hospital.

1. Harmer, D., and Jockes, T.: Proc. Roy. Soc. Med. **12**:187, 1918-1919.

2. Bodin, E., and Lenormond, C.: Recherches sur les poisons produits par l'aspergillus fumigatus, Ann. Inst. Pasteur **26**:371, 1912.

Aspergillus fumigatus is quite familiar as a disease of some birds such as the dove and various other small birds. In them Aspergillus is found in the air passages, and according to some writers produces pneumonia with necrosis of the lung tissue.

In the general population of this country one rarely sees parasites in the nasal passages and their accessory cavities. It is common in India and in people who live in dirt and filth, for example, the Negroes in the South.

In the old literature as far back as 1791 and through the nineteenth century, one finds mention of fungus of the maxillary sinus. The term fungus was used to describe a fungating growth, a spongy mass of morbid granulation tissue or a new growth malignant in character. Hernu [3] and Plaignaud,[4] in 1791, reported cases of fungus of the maxillary sinus. The tissue removed from the antrum was not studied histologically, and cultures were not made. In their cases the history, clinical findings and course of the disease were typical of a malignant growth.

Zarniko,[5] in 1891, reported the first case I could find in the literature of infection of the sinuses due to Aspergillus fumigatus. He stated that, "Aspergillus fumigatus has rarely been found in the nose and never in the sinuses." The patient was white, aged 50, sex not mentioned, and had a chronic suppurative infection of the left maxillary sinus with secondary fungus infection. The chief symptoms were difficulty in breathing through the nose, frontal headache and a thick, greenish, foul-smelling discharge from the nose. Lavage of the antrum showed a mucopurulent discharge and dark brown-grayish material the size of a pea. Microscopic examination showed the presence of a fungus the type of which was believed to be Aspergillus fumigatus, although cultures were not made. The treatment consisted in frequent lavages of the antrum and the internal administration of potassium iodide, from 2 to 4 Gm. a day. The discharge became less, but after sixteen washings the fungus was still found. The patient was not seen again, so the end-result was not mentioned.

Mackenzie,[6] in 1893, reported a case of infection of the maxillary sinus due to Aspergillus mycosis. The patient was a woman, aged 35, who for several years had a chronic, virulent, suppurative infection of

3. Hernu, J. J.: Fongus du sinus maxillaire, abandonné a lui-même; ses ravages; la mort qui les suivit; dissection, J. de chir. **2**:278, 1791.

4. Plaignaud: Observation sur un fongus du sinus maxillaire, J. de chir. **1**: 111, 1791; J. de méd. **87**:244, 1791.

5. Zarniko, C.: Aspergillusmykose der Kieferhöhle, Deutsche med. Wchnschr. **17**:1222, 1891.

6. Mackenzie, J. H.: Preliminary Report on Aspergillus Mycosis of the Antrum Maxillaire, Bull. Johns Hopkins Hosp. **4**:9, 1893.

the right antrum in which Aspergilli had developed in large numbers. The chief symptoms were a chronic nasal discharge, swelling of the right side of the face and over the root of the nose and pain over the right eye and in the right ear. A carious second right upper molar tooth was extracted, the antrum was opened, and the floor was perforated with a drill with return of a large amount of pus. The patient refused further surgical procedures, and for two years the antrum was washed out with various antiseptics which failed to cure the condition. She began to pass threads of a false membrane through the opening of the antrum into the mouth. Sections of the false membrane showed numerous Aspergilli which were believed to be of the fumigatus type. Cultures were not taken. The patient was apparently lost sight of as there is no mention of further treatment and of the end-result.

Harmer,[7] in 1913, reported a case of suppuration of the antrum due to Aspergillus fumigatus. The patient was a woman who had suffered many years with hay fever. She had a persistent discharge of mucus from the right side of the nose and occasional thick, membranous, brownish-yellow cast, which looked like wet blotting paper. The odor was not offensive. She had violent attacks of sneezing, occasional headaches and neuralgic pains around the right eye. The right side of the nose was blocked with edematous looking mucous membrane. The right antrum was dull on transillumination. Her general health was bad, her digestion deranged, and she had the appearance of being poisoned. Her nervous system had suffered, and she could not sleep well at night. The temperature and pulse were normal. The antrum was washed out, and cultures on blood agar plates showed a copious growth of Aspergillus fumigatus in forty-eight hours at 37 C. The mycelium were best seen in unstained films, and the fungus grew best on Sabouraud's maltose agar medium. The antrum was washed out twice a day for over a month with various antiseptic solutions without any improvement. She was then given sodium iodide internally, starting with doses of 2 grains (0.13 Gm.) and rapidly increasing the dosage. In forty-eight hours after this treatment was instituted, a great quantity of the membrane came away. At the end of ten days all discharge had ceased, and after three weeks the antrum was clear on transillumination. The symptoms disappeared, the edema of the nose decreased, and the patient's general health rapidly improved.

Tilley,[8] in 1915, reported some details from five cases of aspergillosis of the maxillary sinus. All five of the patients were women. The chief symptoms were: marked nasal obstruction, sneezing, discharge of

7. Harmer, D.: Suppuration of the Antrum Due to Aspergillus Fumigatus, J. Laryng., Rhin. & Otol. **28**:494 (Sept.) 1913.

8. Tilley, H.: Aspergillosis of the Nasal Accessory Sinuses, J. Laryng., Rhin. & Otol. **30**:145, 1915.

mucoid and slightly mucopurulent material, occasional expulsion of small masses of a whitish-gray, semitranslucent, viscous material and neuralgic pains in the cheek and face and pain in the eye. A Caldwell-Luc operation was done in all five cases. The antrum was found to be filled with a semisolid material, the surface of which was smooth and pale grayish blue; this material was extremely viscous and tenacious and loosely attached to the walls of the sinus from which it was separated with little bleeding. The histologic examination of the material removed from the antrum showed a mucinous material simulating an endothelioma with mucinoid degeneration of the stroma. Mycelial threads and hexagonal crystals were present. Cultures were not made, and the type of Aspergillus was not determined. Tilley stated that after operation there was complete relief from symptoms and no recurrence took place.

REPORT OF A CASE

History.—W. B., a Negress, aged 32, a housewife, of Wilmington, Del., came to Johns Hopkins Hospital on Jan. 24, 1931, complaining of prominence of the left eye, swelling of the left side of the nose and nasal discharge. The family history was irrelevant. The past history revealed dengue fever (?) in 1923 with good recovery, adenoidectomy in 1917 and measles in childhood. She had rheumatism in January, 1930, involving most all of her joints, and had been incapacitated for six months. The patient dated the onset of her present illness to six years before admission to the hospital, when she noticed for the first time that the left eye was slightly more prominent than the right. At that time she was troubled with profuse lacrimation of the left eye and headaches. She was examined in the outpatient department of a northern hospital and was told she had a refractive error. Glasses were prescribed, but the symptoms, headache and lacrimation, persisted; dull aching pain in the left orbital and frontal regions and in the left temple was present most of the day every day and was always worse at night. Occasionally the left eye became red, and not infrequently she had a dull pain in the right eye. For six years she had a chronic nasal discharge from the left side of the nose, frequent colds in the head, left-sided nosebleed and difficulty in breathing through the left side of the nose. Three years ago she noticed a slight swelling of the left side of the nose. Two years ago she had diplopia when looking in any direction except straight ahead. This whole picture gradually became worse. The exophthalmos of the left eye became more pronounced. The vision, however, remained good, and her general health had been good. There was no history of loss of weight.

Physical Examination.—The patient was a well developed and nourished Negro woman, 32 years of age, who was not acutely ill. The temperature was 99 F., the pulse rate 80 and the blood pressure 104 systolic and 70 diastolic. Ophthalmologic findings were as follows: right eye, normal; left eye, definite exophthalmos. There was no bruit or pulsation. The lids closed without difficulty. The movement of the left eyeball was slightly limited in the inferior direction. The palpebral conjunctiva of the lower lid and adjacent bulbar conjunctiva were passively congested. The cornea was clear. The pupils were circular and equal, and reacted to light and accommodation. The anterior chamber was of regular depth. The lens and vitreous were clear. The fundi and the fields were normal. Vision in the right eye was 20/20 —2; in the left eye, 20/70. Examination of the nose

and throat showed the following findings: There appeared to be a thickening of bone below the left orbit and a swelling along the left nasal margin with the maximum point of fulness over the lower portion of the nasal bone, which was definitely tender on pressure. The nasal septum was deflected to the left; the breathing space was very poor on the left side but good on the right. There was a small amount of mucopurulent discharge in the left ethmoidal region; the left inferior turbinate was enlarged. It was impossible to get a satisfactory view of the left ethmoid region due to the marked deflection of the septum. The nasopharynx looked normal. On transillumination, the left frontal sinus and antrum were dark; the right frontal sinus and antrum were clear. There were no enlarged glands at the angles of the jaw or in any of the triangles of the neck. The mouth appeared to be in good condition. The throat and ears were normal. The neurologic examination gave negative results. There was no general glandular enlargement. The heart, lungs, abdomen and extremities were normal.

Laboratory Findings.—The Wassermann reaction of the blood was negative. The hemoglobin (Sahli) was 80 per cent, the red blood cells, 3,290,000, and the

Fig. 1.—Photograph taken before operation, showing swelling along the left lateral margin of the nose, inner angle of the eye and left exophthalmos.

white blood cells, 8,400. The differential count was as follows: polymorphonuclears, 80 per cent; mononuclears and lymphocytes, 20 per cent, and eosinophils, 0 per cent. The urine was normal. The basal metabolic rate was + 11. Roentgenograms of the sinuses, taken on Jan. 24, 1931 (fig. 2), showed marked clouding of the ethmoids, antrum and sphenoid on the left side, and slight clouding of the left frontal sinus, also some clouding of the ethmoids and antrum on the right side. There was an area which suggested erosion of the orbital plate of the ethmoids on the left side; otherwise the orbit was normal. There was no evidence of Paget's disease. Biopsy of tissue removed from the left ethmoidal region (fig. 5) showed that the gross specimen was grayish, slightly transulcent in appearance and firm, but did not have the exact consistency of carcinoma. Its appearance was suggestive of fibrous tissue rather than of tumor. Microscopic examination showed dense fibrous tissue with numerous giant cells. They occurred singly and were not included in definite tubercle formation. One could see in the giant cells bodies having a doubly refractile membrane around them. The doubly refractile bodies were seen in most of the giant cells. Special Gram stain showed mycelial bodies running through the tissue. In places they were branched (fig. 4). A pathologic diagnosis of fungus infection was made. There was no evidence of

tumor in the sections. However, it was suggested that the fungus infection might be a secondary invader and the underlying primary lesion a tumor. A culture was taken from the left ethmoidal region at the time of the biopsy and put on Sabouraud's maltose agar medium. A growth occurred in ten days, which was first greenish in appearance but later turned to a brownish black (fig. 3). Under the microscope one could see mycelial filaments with an enormous number of spores. The mycelial filaments were more or less ramified; frequently they were branched and the condiophorehyphia were much thicker than the mycelial tubes. The conidia were round. Culturally and microscopically, they were typical of Aspergillus fumigatus. Transfer cultures grew out in forty-eight hours and were isolated two or three times.

Diagnosis.—Clinically, the diagnosis was indeed puzzling. The various impressions were mucocele, new growth, tumor involving the orbit, gumma, Paget's disease and osteitis. On the basis of the results of the pathologic and biologic

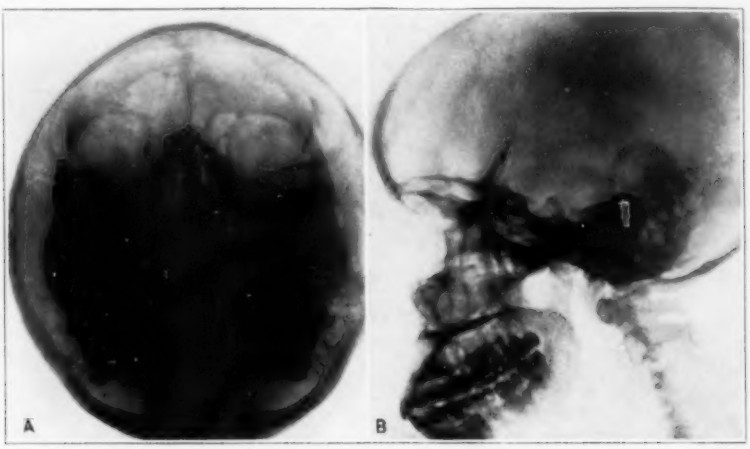

Fig. 2.—Roentgenograms of the sinuses taken on Jan. 24, 1931. *A*, anterior-posterior view, showing clouding of the anterior ethmoids and antrum bilateral, particularly on the left, and slight clouding of the left frontal sinus. There is an area which suggests erosion of the orbital plate of the ethmoids on the left side. *B*, lateral view, showing clouding of the posterior ethmoids and sphenoid on the left.

examinations and the negative Wassermann reaction, a diagnosis was made of infection due to Aspergillus of the fumigatus type involving the ethmoid, maxillary and sphenoid sinuses and the orbit on the left side.

Treatment and Course.—An exploratory puncture of the left antrum beneath the inferior turbinate was made. Neither air nor solution could be blown through the cannula. The trochar felt as if it passed through solid tissue. The nasal septum, which was markedly deflected to the left, had to be straightened in order to get a good view of the ethmoidal region. This was done under local anesthesia on Feb. 10, 1931. The anatomic landmarks on the left were obliterated and the middle turbinate was not seen, but the region of the middle turbinate, ethmoids and middle meatus was filled with tissue which had a grayish-white appearance, was firm and bled easily. There was a moderate amount of purulent discharge in this region. All of the ethmoidal cells were filled with this growth. A biopsy was

made and a culture taken. The pathologic and biologic observations have been previously described. Even after careful cocainization, the ethmoidal region never did seem to become totally insensitive. On account of this and the troublesome bleeding, nothing more was done at this sitting. The incision in the septum was closed with black silk sutures, the left side of the nose was packed with gauze, and the patient was sent back to the ward. The nasal pack was removed the following day.

The patient was then given large quantities of potassium iodide; she received 100 grains (6.5 Gm.) of a saturated solution of potassium iodide by mouth daily, the dose being gradually increased until she was receiving 200 grains (13 Gm.) daily. The following two weeks she complained of almost constant headache, pain in and around the left eye and over the left side of the nose and cheek. The left eye and left side of the nose became more prominent and sore to the touch. The left eye felt weak. The temperature ranged between normal and 101 F.; the

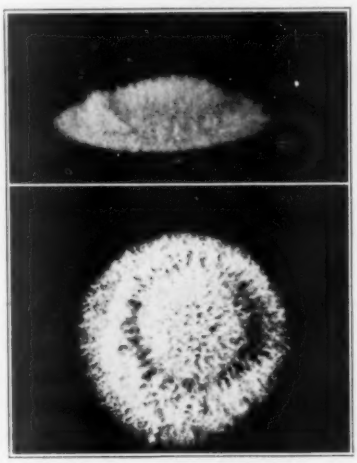

Fig. 3.—Culture from the left ethmoids on Sabouraud's maltose agar medium. A growth occurred in ten days, which was first greenish in appearance but later turned to a brownish black. Transfer cultures grew out in forty-eight hours, and were isolated two or three times.

pulse, between 90 and 120. The potassium iodide began to bring on nausea and vomiting and was discontinued for a few days.

On March 3, 1931, an exploratory operation on the left antrum was done through the canine fossa under avertin-ether anesthesia. The bone in the region of the canine fossa was hard and white, and the outer aspect showed no evidence of necrosis. In using the perforator, hard bone was encountered for a short distance, and then it seemed to enter dense fibrous tissue, which was almost as hard as bone. The left antrum was filled with this tissue. No extensive removal of bone and tissue was attempted at this operation. Bone and tissue were removed for microscopic study, cultures were taken, and a large counter opening was made into the nose beneath the inferior turbinate. A protective gauze pack was placed in the antrum and its end brought out through the nose. The incision beneath the lip was closed with interrupted sutures of 00 plain catgut. The pack in the antrum was removed the following day. It was thought at the time of this operation that

there might be a growth of some kind, the fungus infection merely being a secondary invader. The gross and microscopic appearance of the tissue removed from the left antrum was the same as that removed from the left ethmoidal region, and none of the sections showed anything to suggest a tumor or new growth. The wound beneath the lip soon broke down, and there was a foul smelling discharge. The patient was given smaller doses of a saturated solution of potassium iodide in water, 3 cc. three times a day, which was increased 0.1 cc. per dose a day. This was given by rectum in order to lessen the nausea. Cultures taken from the left antrum three weeks after starting treatment with iodides were negative for fungi. The two bicuspid and first molar teeth in the left upper jaw became loose. The left antrum was kept as clean as possible with irrigations of potassium permanganate. The tissues around the wound in the canine fossa became soft and necrotic, and an opening developed between the left antrum and the mouth. The patient

Fig. 4.—Photomicrograph of tissue removed from the left ethmoidal region (high magnification), showing mycelium. The mycelial filaments were more or less ramified, the breadth varying between 2 and 3 microns; frequently they were branched. The condiophorehyphia are much thicker than the mycelial tube, being on the average of about 5 microns in breath. The conidia are roundish. Mycelium were seen best in unstained films. Fungi of the genus Aspergillus, when growing parasitically in the tissues, often lose their characteristics; the typical fructifications are absent, and only mycelial threads and roundish or oval yeastlike bodies are seen. Special Gram stain was used.

continued to complain of headache and pain in the left eye and cheek. The exophthalmos became more pronounced, and the left eye became red with a marked conjunctivitis. The temperature ranged between normal and 102 F.; the pulse between 100 and 120. The patient appeared toxic and was very nervous and irritable, and her digestion was deranged.

On March 18, 1931, under avertin-ether anesthesia, a nasal operation on the ethmoids and sphenoid and a radical operation on the antrum were done on the

left side. As the two bicuspid and first molar teeth were very loose, they were removed. Large masses of dense, hard, fibrous, grayish-white tissue were removed from the left antrum. The surrounding bone, particularly in the floor of the antrum, the nasal wall and parts of the lateral and anterior walls, was soft and came away; it broke off much like wet blotting paper. All of the growth was thoroughly removed from the antrum, and all the surrounding soft bone was removed until hard, normal looking bone was encountered. No defect was seen in the floor of the orbit. The entire lateral wall of the nose was removed, thus making a huge opening into the nasal cavity. The ethmoids were thoroughly cleaned out and found to contain the same sort of growth as in the antrum. The left sphenoid was opened and was found filled with pus and the same sort of growth as in the ethmoids and antrum. The sphenoid was widely opened and as much of the growth as it was possible to take out with safety was removed. The growth extended up around the region of the cribriform plate and as much was removed as possible within the limits of safety. Numerous specimens of the tissue were sent to the laboratory for microscopic study, and cultures were again taken. A protective gauze pack was placed in the antrum and its end brought out through the nose. The opening into the mouth was closed with interrupted sutures of 00 plain catgut. The antral pack was removed the next day.

The following week the temperature ranged between normal and 100 F.; the pulse, between 100 and 120. The wound between the mouth and left antrum soon broke down again with a foul smelling discharge and was kept clean with irrigations of potassium permanganate. The cultures taken from the left antrum, ethmoids, sphenoid and canine fossa were negative for fungi. It is interesting to note that the fungi were not demonstrated in the tissues removed from these regions and the teeth, a special modification of the Gram stain being used, whereas before treatment was started with the iodides the tissue in the ethmoidal region was loaded with fungi which could easily be demonstrated by means of a special stain. The hemoglobin decreased to 62 per cent. The patient was put on a high caloric diet containing 200 Gm. of liver.

Three weeks following the last operation a stomatitis developed—small superficial ulcerations all around the edge of the tongue with sloughing of epithelial tissue. Cultures failed to show the presence of fungi. It was difficult for the patient to take nourishment by mouth on account of extreme pain. Nutrient enemas were given. The stomatitis cleared in a few days with the use of potassium chlorate as a mouth wash. Slight headache continued, the left exophthalmos became a little more pronounced, and the left eye was painful and red, with a conjunctivitis and inflammation of the lacrimal duct, but this improved with the use of irrigations of boric acid. Because of such good vision in the left eye, it did not seem justifiable to sacrifice the eye.

There was a slow but gradual improvement in the patient's local and general condition. The toxemia lessened, and she was not nearly so nervous and irritable. She was discharged on April 29, 1931, after three and a half months' stay in the hospital.

When seen a month later she felt and looked much improved but complained of slight pain over the top of the head. Fluids taken into the mouth would run out of the left nostril. There was a small fistulous opening through the canine fossa between the left antrum and the mouth. The nose and sinuses were clean and free from discharge. The exophthalmos was less marked. There was no pain in the eye or cheek. There was a good breathing space through the nose. The conjunctivitis and inflammation of the lacrimal duct had cleared. Seven weeks later the patient was seen again, and cultures were taken from the nose and sinuses. They

were negative for fungi. When she was seen three months later she had gained 14 pounds (6.4 Kg.) in weight and felt better than she had for several years. She had no headache or pain in the left eye, nose, cheek or jaw. There was no diplopia. Her vision was good. The external swelling of the left side of the nose and exophthalmos were scarcely noticeable. The eyegrounds looked normal. There were no signs of inflammation of the conjunctiva or of the lacrimal duct. The

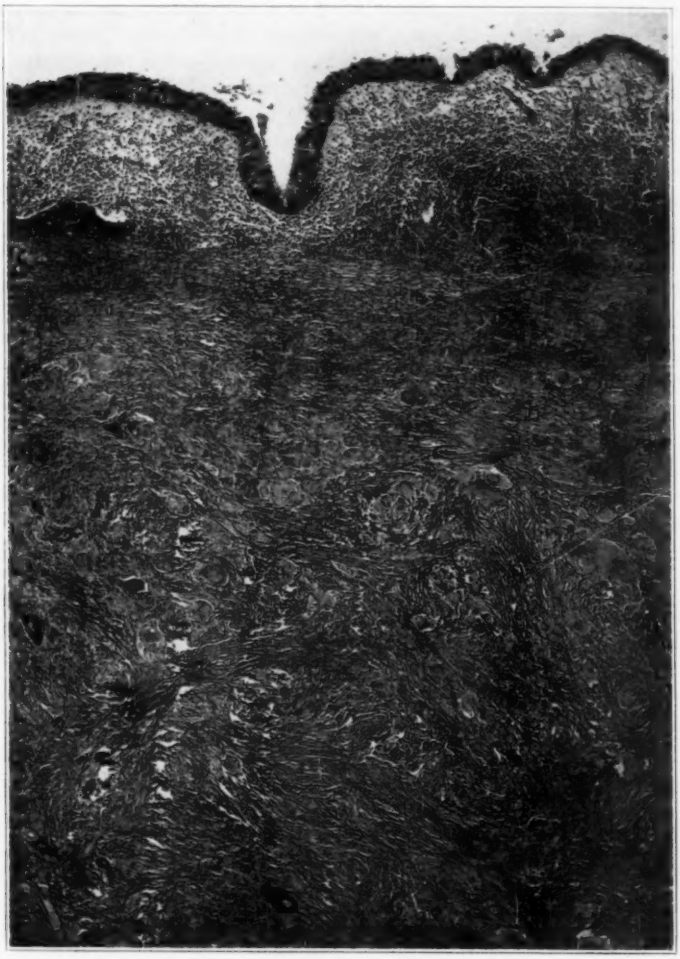

Fig. 5.—Photomicrograph of tissue removed from the left ethmoidal region, showing dense fibrous tissue which in places is almost hyalinized, and numerous giant cells. The giant cells resemble quite strikingly foreign body giant cells. They occur singly and are not included in definite tubercle formation; there is merely a giant cell surrounded by fibrous tissue. Under the microscope one could eee oval-like bodies in most of the giant cells which had a doubly refractile membrane around them. Special Gram stain showed numerous mycelia throughout the tissue. In places they were branched. The epithelium is intact and still ciliated in places. The submucosa is edematous and contains mononuclear cells, most of which are plasma cells with some eosinophils.

movement of the left eye was still slightly limited in the outward and downward direction. A small fistulous opening through the canine fossa from the left antrum into the mouth remained. The nose and sinuses were clear and free from discharge. The hard palate looked normal. The patient did not look or feel toxic. She was no longer nervous and irritable, and her digestion was good. The temperature was normal. She was again admitted to the hospital for closure of the fistula opening between the left antrum and the mouth, which was done under avertin-ether anesthesia.

COMMENT

This case is interesting because of the rarity of the condition and because of the extension of the fungous infection, involving not only the maxillary sinus but the ethmoids, sphenoid and orbit. In the cases reported in the literature by Zarniko, Mackenzie, Harmer and Tilley, the maxillary sinus alone was involved. It is also interesting to note that in the eight previously reported cases of fungus infection of the sinuses, seven were in women, in one the sex was not mentioned, and the case reported here was in a woman. The mode of infection is not definitely known. The course of the disease is slow and progressive. In the present case the disease probably existed for six years unrecognized. Clinically, the symptoms are those of a chronic suppurative sinusitis with toxic manifestations. The disease may easily be confounded with an ordinary chronic suppurative sinusitis, mucocele and new growth. Only by careful biologic and pathologic studies can a diagnosis be definitely made. The treatment that was found efficacious in this case was the internal administration of large quantities of a saturated solution of potassium iodide and thorough surgical removal of the growth. After one year the patient was free from symptoms, and there has been no recurrence.

DUODENAL DIVERTICULA

AN ANATOMIC STUDY, WITH NOTES ON THE ETIOLOGIC RÔLE PLAYED BY DYSTOPIA OF PANCREATIC TISSUE

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Among the historic considerations that we have reviewed on diverticulum of the duodenum, Chomel,[1] in 1710, is credited with having first described the condition. It is believed by most writers, however, that Chomel's case was probably one of dilatation of the ampulla of Vater which contained twenty-two stones. Case,[2] in 1913, first diagnosed diverticula of the duodenum roentgenologically. Forssell and Key,[3] in 1915, diagnosed a case in which the diverticulum was the first to be removed surgically. Linsmayer,[4] in 1919, reviewed the subject from an anatomic standpoint, as did Odgers,[5] Grant,[6] Herbst,[7] Nagel[8] and others, and Spriggs and Marxer,[9] in 1925, gave an excellent summary from a roentgenologic point of view.

From the Division of Medicine, the Mayo Clinic. The work was done in the Section on Pathologic Anatomy.

Abridgment of thesis submitted by Dr. Mueller to the Faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Master of Science in Medicine, October, 1931.

1. Chomel, quoted by Buschi: Virchows Arch. f. path. Anat. **206**:121 (Oct. 13) 1911.

2. Case, J. T.: Diverticula of Small Intestine, Other Than Meckel's Diverticula, J. A. M. A. **75**:1463 (Nov. 27) 1920.

3. Forssell, Gösta, and Key, Einar: Divertikel å pars descendens duodeni diagnosticeradt medels röntgenundersökning och operativt aflägsnadt, Nord. med. Ark. **48**:1, 1915; Ein Divertikel der Pars descendens duodeni mittels Röntgenuntersuchung diagnostiziert und operativ entfernt, ibid. **48**:15, 1915.

4. Linsmayer, Heinrich: Ueber Duodenaldivertikel, Verhandl. d. deutsch. path. Gesellsch. **17-18**:445, 1919.

5. Odgers, P. N. B.: Duodenal Diverticulosis, Brit. J. Surg. **17**:592 (April) 1930.

6. Grant, J. C. R.: Duodenal Diverticula, J. Anat. **57**:357 (July) 1923.

7. Herbst, W. P.: Diverticula of the Duodenum, Minnesota Med. **10**:364 (June) 1927.

8. Nagel, G. W.: Unusual Conditions in the Duodenum and Their Significance, Arch. Surg. **11**:529 (Oct.) 1925.

9. Spriggs, E. I., and Marxer, O. A.: Intestinal Diverticula, Quart. J. Med. **19**:1 (Oct.) 1925.

In recent years frequent references have been made to the symptoms and complications which may be caused by duodenal diverticula. This is probably due to the increased significance ascribed to them since they began to be diagnosed by means of the roentgen rays. Scott,[10] in a recent publication, stated that all duodenal diverticula probably eventually cause symptoms, and that they are one of the causes of the chronic condition of the abdomen, together with that of duodenal ileus and splenic drag.

REVIEW OF LITERATURE

Following Chomel, Morgagni,[11] in 1761, described the next case of diverticulum of the duodenum. The nature of the diverticulum is questionable, although Buschi,[12] in his review, mentioned that Morgagni had recorded the first true cause of duodenal diverticulum. Fleischmann,[13] in 1815, reported 3 typical cases. Subsequently, there were occasional reports of cases from postmortem material.[14] Harley's report, in 1857, was the first to appear in English literature. He described a diverticulum containing a large gallstone, and its true nature is questionable. Cruveilhier,[15] in 1849, when writing on diverticula of the gastro-intestinal tract, described pouches in the esophagus and colon, and stated that diverticula did not occur between these two points. Perry and Shaw,[16] in 1893, gave the most detailed report which had appeared since Fleischmann's report; they reviewed 10 cases from the postmortem records of Guy's Hospital from 1826 to 1892, and 4 cases from other sources. Following this, the most noteworthy considerations and reports of cases came from Rolleston and Fenton[17] in 1900,

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10. Scott, S. G.: Diverticula of the Duodenum, Brit. M. J. **1**:346 (Feb. 28) 1931.

11. Morgagni, quoted by Buschi.[12]

12. Buschi, Giuseppe: Beitrag zur Untersuchung der Duodenaldivertikel, Virchows Arch. f. path. Anat. **206**:121 (Oct. 13) 1911.

13. Fleischmann, quoted by Buschi.[12]

14. Habershon, S. O., quoted by Spriggs and Marxer.[9] Harley, George: Specimen of Hepatic-Intestinal Calculus, Tr. Path. Soc. London **8**:235, 1857. Klebs, quoted by Hansemann: Virchows Arch. f. path. Anat. **144**:400 (May 6) 1896. Moore, Norman: Multiple Diverticula of the Small Intestine with Congenital Stricture of the Duodenum, Tr. Path. Soc. London **35**:202, 1884. Roth, M.: Ueber Divertikelbildung am Duodenum, Virchows Arch. f. path. Anat. **56**:197 (Nov. 1) 1872.

15. Cruveilhier, quoted by Spriggs and Marxer.[9]

16. Perry, E. C., and Shaw, T. E.: On Diseases of the Duodenum, Guy's Hosp. Rep. **50**:171, 1893.

17. Rolleston, H. D., and Fenton, W. J.: Two Anomalous Forms of Duodenal Pouches, J. Anat. & Physiol. **35**:110 (Oct.) 1900.

Fischer [18] in 1901, Voigtel [19] in 1904, Bassett [20] in 1907, Gordinier and Sampson [21] in 1906, Keith [22] in 1910, Baldwin [23] in 1911, Buschi in 1911, Bauer [24] in 1912, Davis [25] in 1913 and Wilkie [26] in 1913. Thus far, all cases reported had been found at necropsy. Since the first roentgenologic diagnosis of duodenal diverticulum in 1913, many references to it have been found in the literature, and diverticula are now frequently diagnosed roentgenologically.[27] Numerous instances are reported of removing the diverticulum surgically.[28]

Hypotheses with regard to etiology are as follows: Fleischmann, Keith [29] and Roth stated their belief that herniation of the duodenal mucosa occurred at the points where the duodenal musculature was weakened by penetration of the bile and pancreatic ducts. Letulle and Nattan-Larrier,[30] however, tried to show that the musculature about the duodenal papillae is stronger than that in other regions of the duodenum. Klebs, in 1869, was the first to call attention to the part played by blood vessels in the formation of diverticula of the intestine

18. Fischer, M. H.: False Diverticula of the Intestine, J. Exper. Med. **5**:333 (Jan. 15) 1901.

19. Voigtel, quoted by Spriggs and Marxer.[9]

20. Bassett, V. H.: Duodenal Diverticula, with Especial Reference to Diverticula Associated with the Pancreatic and Biliary Ducts, Tr. Chicago Path. Soc. **7**:83 (Dec.) 1907.

21. Gordinier, H. C., and Sampson, J. A.: Diverticulitis (not Meckel's) Causing Intestinal Obstruction, J. A. M. A. **46**:1585 (May 26) 1906.

22. Keith, Arthur: A Demonstration on Diverticula of the Alimentary Tract of Congenital or of Obscure Origin, Brit. M. J. **1**:376 (Feb. 12) 1910.

23. Baldwin, W. M.: Duodenal Diverticula in Man, Anat. Rec. **5**:121 (March) 1911.

24. Bauer, Theodor: Ueber das Duodenaldivertikel, Wien. klin. Wchnschr. **25**: 879 (June 6) 1912.

25. Davis, N. S.: Diverticula of the Duodenum, Tr. Chicago Path. Soc. **9**: 1 (Feb.) 1913.

26. Wilkie, D. P. D.: Duodenal Diverticula and Duplicature of the Duodenal Wall, Edinburgh M. J. **11**:219 (Sept.) 1913.

27. Scott.[10] Carman, R. D.: The Roentgenologic Diagnosis of Duodenal Ulcer, Am. J. Roentgenol. **3**:252 (May) 1916. Lockwood, A. L.: Duodenal Diverticula, Tr. A. Res. and ex-Res. Physicans, Mayo Clin. **10**:54, 1929.

28. Forssell.[3] Basch, Seymour: Diverticulum of the Duodenum, with a Report of a Case Diagnosed During Life and Successfully Operated on, Am. J. M. Sc. **153**:833 (June) 1917. Downes, W. A.: A Duodenal Diverticula, Ann. Surg. **76**: 43 (July) 1922. Lewis, Dean: A Duodenal Diverticulum, J. A. M. A. **76**:783 (March 19) 1921. Maclean, N. J.: Diverticulum of the Duodenum, Surg., Gynec. & Obst. **37**:6 (July) 1923. Siegrist, Hans: Ein Beitrag zur Kasuistik der Duodenaldivertikel, Cor.-Bl. f. schweiz. Aerzte **49**:47 (Jan. 4) 1919.

29. Keith, Arthur: The Nature and Anatomy of Enteroptosis (Glénard's Disease), Lancet **1**:631 (March 7) 1903.

30. Letulle, Maurice, and Nattan-Larrier: Région vatérienne du duodénum et ampoule de Vater, Bull. et mém. Soc. anat. de Paris **12**:491 (June) 1898.

other than duodenal, and this idea has been reemphasized by Hansemann,[31] Graser [32] and Fischer. Keith also advocated the idea that traction exerted at the side of the common bile duct by ptosis of abdominal viscera and increased intra-intestinal pressure might, in certain cases, give rise to these diverticula. Roth, who was probably the first to advocate the idea of traction, believed that the pull on the duodenum might be exerted by a pancreas which was receding, owing to atrophy. Chomel and Harley concluded that traction might be exerted by scar tissue, and more recently, Davis stated that the superior and inferior pancreatic-duodenal arteries may cause the necessary amount of traction in cachectic subjects. Opposed to the traction hypothesis were Buschi and Jach,[33] who argued that if pull were exerted it would not be confined to isolated small areas of the duodenum, as would necessarily be the case in the formation of duodenal diverticula. They also argued that if an atrophic, receding pancreas were to exert the necessary traction, dense fibrous tissue would have to be interposed between it and the duodenal wall, and this is never found.

Various writers have favored the congenital origin of these structures, among them Buschi, Gandy,[34] Letulle,[35] Marie [36] and Wilkie. Buschi argued that the duodenum shows a predisposition to congenital anomalies because it is the portion of the intestinal tract which undergoes the greatest changes during fetal development. He quoted Shaw, who found a diverticulum associated with duodenal stenosis in an infant. He also quoted Lewis and Thyng's [37] demonstration of epithelial outgrowths from the duodenum in early human, pig and rabbit embryos, and the association of diverticula with accessory pancreatic tissue. Other arguments in favor of the congenital origin are the facts that duodenal diverticula may be accompanied by other gastro-intestinal diverticula,[26] and that they are seldom associated with inflammation or dilatation of the duodenum itself.[9]

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31. Hansemann, David: Ueber die Entstehung falscher Darmdivertikel, Virchows Arch. f. path. Anat. **144**:400 (May 6) 1896.

32. Graser, Ernst: Ueber multiple falsche Darmdivertikel in der Flexura sigmoidea, München. med. Wchnschr. **46**:721 (May 30) 1899.

33. Jach, quoted by Buschi.[12]

34. Gandy, M. C.: Diverticule duodénal congénital, Bull. et mém. Soc. anat. de Paris **2**:691 (July) 1900.

35. Letulle, Maurice: Malformations duodénales. Diverticules périvatériens, Bull. et mém. Soc. anat. de Paris **12**:807 (Dec.) 1898.

36. Marie, M. R.: Diverticules duodénaux périvatériens, Bull. et mém. Soc. anat. de Paris **1**:982, 1899.

37. Lewis, F. T., and Thyng, F. W.: The Regular Occurrence of Intestinal Diverticula in Embryos of the Pig, Rabbit, and Man, Am. J. Anat. **7**:505 (Feb. 29) 1908.

Increasing consideration has been accorded the idea that a congenital muscular defect forms a *locus minoris resistentiae* which is the basis for the later development of intestinal diverticula.[38] The localized weakness is said to be due either to penetration of the bile and pancreatic ducts, or of blood vessels as previously stated, or simply to pathologic changes in the musculature. Andrews,[39] Bassett, Opie,[40] Ribbert,[41] and others made brief statements as to the influence of accessory pancreatic tissue in weakening the wall of the bowel, whereas Hansemann reasserted that traction on the accessory pancreas, as described by Neumann and Nauwerck,[42] was a factor in the production of diverticula of the small bowel. Cases of intestinal diverticula other than duodenal, associated with an accessory pancreas, have been reported by Hanau,[43] Klob,[44] Weichselbaum,[45] Zenker[46] and others, and Gardiner[47] reported a case of a congenital hour-glass stomach combined with an accessory pancreas. Linsmayer, in 1919, to whose paper we wish to draw particular attention, emphasized the influence of the pancreas above all other factors. In addition to the fact that he always found the diverticula in close association with the pancreas, as have other observers, he stated that in the smallest diverticula, which were not fully formed, he found small lobules of pancreas between the muscle bundles; in 3 duodenums without diverticula he found pancreatic tissue embedded in the muscularis externa. He stated that the development of a duodenal diverticulum is dependent on the displacement of pancreatic tissue in the wall of the intestine, which tissue produces a circumscribed *locus minoris resistentiae,* and that duodenal diverticula "are congenital only so far as the predisposition to them occurs, due to dystopia of pancreatic tissue arising in earliest fetal life." In this connection it may be noted that Zenker, in 1861, found an accessory pancreas in 5 cases, lying within the wall of the intestine; 1 of these lay near the tip of a finger-like true diverticulum, 5 cm. long.

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38. Nagel.[8] Mayo, W. J.; Wilson, L. B., and Giffin, H. Z.: Acquired Diverticulitis of the Large Intestine, Surg., Gynec. & Obst. **5**:8 (July) 1907.

39. Andrews, E. W.: Duodenal Diverticula, J. A. M. A. **77**:1309 (Oct. 22) 1921.

40. Opie, E. L.: Diseases of the Pancreas, ed. 2, Philadelphia, J. B. Lippincott Company, 1910.

41. Ribbert, Hugo: Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie, ed. 7, Leipzig, F. C. W. Vogel, 1920, p. 489.

42. Nauwerck, C.: Ein Nebenpankreas, Beitr. z. path. Anat. u. z. allg. Path. **12**:29, 1893.

43. Hanau, quoted by Lewis,[48] p. 441.

44. Klob, quoted by Opie.[40]

45. Weichselbaum, A., quoted by Lewis.[48]

46. Zenker, F. A.: Nebenpancreas in der Darmwand, Virchows Arch. f. path. Anat. **21**:369, 1861.

47. Gardiner, J. P.: A Case of Congenital Hour-Glass Stomach with Accessory Pancreas, J. A. M. A. **49**:1598 (Nov. 9) 1907.

In an attempt to obtain some definite information on the fetal development of the pancreas and duodenum, a brief review of studies on the embryology of these organs was made. Lewis [48] stated that a pancreas both dorsal and ventral has been found in 3 and 4 mm. embryos, and that an accessory pancreas is most frequently connected with the stomach, but also occurs in the duodenum, jejunum and ileum, and "has been frequently found at the apex of a true diverticulum." Lewis and Thyng found epithelial nobs in human, as well as in pig and rabbit embryos, which were interpreted as epithelial diverticula. Helly [49] described the epithelial outgrowth from the duodenum, penetrating the mesoblastic portion of the wall of the intestine.

The development of the well differentiated muscle layers occurs at a much later date than that of the epithelial elements, according to Lewis,[48] who stated that the longitudinal muscle of the duodenum becomes distinct at the 75 mm. stage. He gave no time for the development of the circular muscle, which is probably developed somewhat earlier than the longitudinal; one of us (Dr. Horton [50]) observed that the circular muscle layer in the stomach and duodenum is well developed in a 41 mm. human embryo.

With regard to the form and position of the pancreas in some of the lower vertebrates, Oppel [51] quoted Maas [52] as having found a pancreas-like organ embedded in the serosa of the wall of the intestine in myxine glutinosa, one of the cyclostomas, and Brachet [53] found a ring-shaped mass of cells around the circumference of the midgut beneath the epithelium, in ammocetes. In Salamandra maculata, the longitudinal muscle partially surrounds the pancreas for a short distance.

The clinical significance of duodenal diverticula is still debatable. Baldwin, Bauer, Davis, Fischer and Wilkie reported isolated cases of duodenal diverticula which were believed to have caused obstruction and even death. Occasional cases of perforation have also been reported.[54]

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48. Lewis, F. T.: The Development of the Small Intestine, the Large Intestine, the Liver, and the Pancreas, in Keibel, Franz, and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, vol. 2, p. 381.

49. Helly, Konrad: Zur Pankreasentwickelung der Säugetiere, Arch. f. mikr. Anat. **57**:271, 1901.

50. Horton, B. T.: Pyloric Musculature, with Special Reference to Pyloric Block, Am. J. Anat. **41**:197 (May) 1928.

51. Oppel, Albert: Lehrbuch der vergleichenden mikroskopischen Anatomie der Wirbeltiere, Jena, Gustav Fischer, 1900, vols. 2 and 3.

52. Maas, Otto, quoted by Oppel,[51] vol. 3, p. 825.

53. Brachet, A., quoted by Oppel,[51] vol. 3, p. 829.

54. Lucinian, J. H.: Diverticulum of the Duodenum Perforated into the Pancreas, Am. J. Roentgenol. **24**:684 (Dec.) 1930.

Cole and Roberts,[55] in 1920, suggested that possibly primary carcinoma of the duodenum arises from the pancreatic tissue which is so commonly found in the walls of duodenal diverticula. Confirmation of this statement has not been found in the literature. The association of diverticula with duodenal ulcer and cholecystitis has been emphasized by some writers.[56] However, Robertson and Hargis [57] did not mention duodenal diverticula in their report, in which they emphasized shortening of the distance between the ampulla and pylorus in cases of duodenal ulcer.

MATERIALS AND RESULTS

The material for this study was taken from the postmortem records and specimens of the Mayo Clinic, up to June 1, 1931. The records were first reviewed, and the description was noted of all structures diagnosed as duodenal diverticula. These specimens were then studied, and the final analysis excluded those so-called diverticula which had formed as results of duodenal ulcerations. Pouchings and deep beds of scar tissue in the first portion of the duodenum were frequently designated as duodenal diverticula in the older records, and occasionally even as late as 1923. However, in recent years the pouchings in the region of the pylorus were designated as such, and the deep ulcer-beds were recorded as ulcers. The final material analyzed consisted, therefore, only of those duodenal diverticula which, in the literature, are commonly designated as "false" or "primary" diverticula or as "mucosal hernias," and which we will refer to as "duodenal diverticula."

The total number of duodenums in which diverticula were found was 122, the first one having been recorded in 1913. The total number of diverticula of the duodenum in these 122 cases was 145. One hundred and eight of these occurred singly, whereas in 14 of the duodenums examined 2 or more diverticula were found. In other words, 88.53 per cent of the duodenal diverticula in this series occurred singly, whereas 11.47 per cent occurred as multiple diverticula. Of these multiple diverticula 2 occurred in 8 cases, 3 diverticula in 4 cases, and 4 and 5 diverticula in 1 case each; thus greater multiplicity occurs only rarely. Among those listed as single diverticula are included 7 which were bilocular; that is, they had a single opening but with a subdivision of the sac into two parts by a septum.

A large percentage of the diverticula occurred in close relationship to the major and minor duodenal papillae. The position of the diverticula

55. Cole, L. G., and Roberts, Dudley: Diverticula of Duodenum: Their Clinical and Roentgenological Recognition, Surg., Gynec. & Obst. **31**:376 (Oct.) 1920.

56. Perry and Shaw.[16] Wilkie.[26]

57. Robertson, H. E., and Hargis, E. H.: Duodenal Ulcer: An Anatomic Study, M. Clin. North America **8**:1065 (Jan.) 1925.

with relation to the papilla of Vater was determined either from the protocol or by examination of the specimen in 141 instances. The position in 4 of the 145 cases was not ascertainable. In 82 cases (58.15 per cent of 141), the diverticulum was closely associated with the papilla of Vater (figs. 1, 2 and 3). Thirty-six (25.53 per cent) of the diverticula occurred proximal to the papilla of Vater, and most of these were apparently adjacent to the minor papilla, which frequently contains pancreatic tissue besides the duct of Santorini. Thus, 83.68 per cent of the diverticula occurred in association with pancreatic ducts. Twenty-three (16.31 per cent) of the diverticula were situated distal to the papilla. None was found in the first portion of the duodenum. These statements apply to the position with relation to the long axis of the duodenum. An additional and very important point to be emphasized is the position with relation to the circumference of the duodenum. In every instance, the orifice of the diverticulum occurred on the concave or pancreatic border of the duodenum, and the sac either penetrated or lay against the pancreas. The smaller sacs were buried in the pancreas. Those that were larger were in contact with the pancreas only at the neck of the sac, with the fundus covered by a small amount of areolar tissue, and lying either dorsal or ventral to the duodenum and the pancreatic head (fig. 1 *A*).

The size of the diverticula varied considerably. The diameter of the orifice, which is usually circular, and the depth of the sac were the two measurements most frequently recorded. The smallest diverticulum consisted of a tiny, blind tract which had burrowed down along the intramural portion of the common bile duct for 0.3 cm., its orifice being only 0.2 cm. in diameter and situated at the base of the papilla. The largest diverticulum measured 5.0 cm. in diameter at the orifice, and the sac was 7.0 cm. in depth and lay superior and slightly dorsal to the head of the pancreas. These two cases represent the extremes in size. The average diameter of the orifices was 1.15 cm., the diameters most frequently measuring between 1.0 and 2.0 cm. The average depth of all the sacs measured was 2.01 cm. The depth of the sac varied more than did the diameter of the orifice, but the majority were between 1 and 2.5 cm.

Most of the sacs had orifices which were smaller in diameter than the widest diameter of the deeper portions of the sac; that is, they were flask-shaped (fig. 1 *A*). In a few instances the mucosa at the rim of the orifice was thickened and formed an overhanging thick ridge. In those diverticula which were very small or which, though broad, were still in an early stage, this flasklike characteristic was lacking; the orifice was as large as, or larger, than the widest portion of the depressed area (figs. 1 *B*, 2 and 3).

Microscopically, the duodenal diverticula were found to be primarily mucosal structures, the majority having no muscle layers in their walls.

In those which we believe to be early diverticula, the muscle layers of the duodenum extended for a variable distance down into the wall of the pouch, but were practically discontinued near the fundus of the pouch (figs. 2, 3 and 4). The fundus was thus composed only of mucosa, submucosa and a small amount of connective tissue resting directly on the pancreas, with occasional small, smooth muscle bundles present in the connective tissue at the fundus. In those early diverticula which were situated immediately adjacent to the papilla of Vater or to the

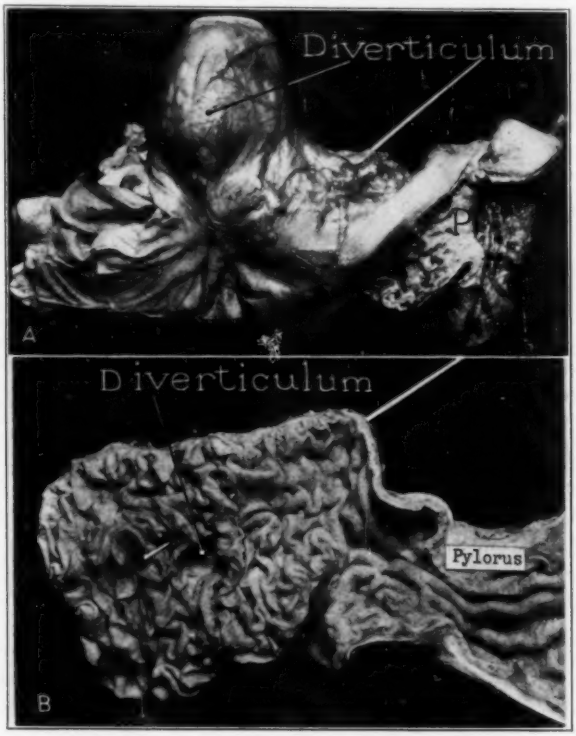

Fig. 1.—*A*, external aspect of large diverticulum from a man, aged 77. The sac is 5 cm. deep. *P* indicates pylorus. *B*, early diverticulum from a man, aged 61. The probe is in the common bile duct, and projects through the ampulla of Vater. A broad, shallow diverticulum and one of a small, shallow type may be observed in this and in figures 2 and 3.

minor papilla, the duct musculature was present on one side of the pouch (figs. 2 and 3). In all of the large and older diverticula, the muscle layers of the duodenal wall stopped quite abruptly at the orifice, leaving the wall devoid of muscle or practically so. Occasionally a few small muscle bundles were found deep at the fundus of the sac, but these were never prominent and had to be searched for carefully. In

two fairly early diverticula, which we believe were destined to be bilocular had the patients lived longer, there were fairly large masses of muscle between the mucosa and the pancreas at the fundus of the pouch. These masses caused low projections upward into the sacs, forming the beginnings of double sacs. The muscularis mucosa was intact in all instances and occasionally seemed to be hypertrophied.

The mucosa in the earlier diverticula was thicker and contained more glands and villi than in the older sacs (figs. 2, 3 and 4). Brunner's

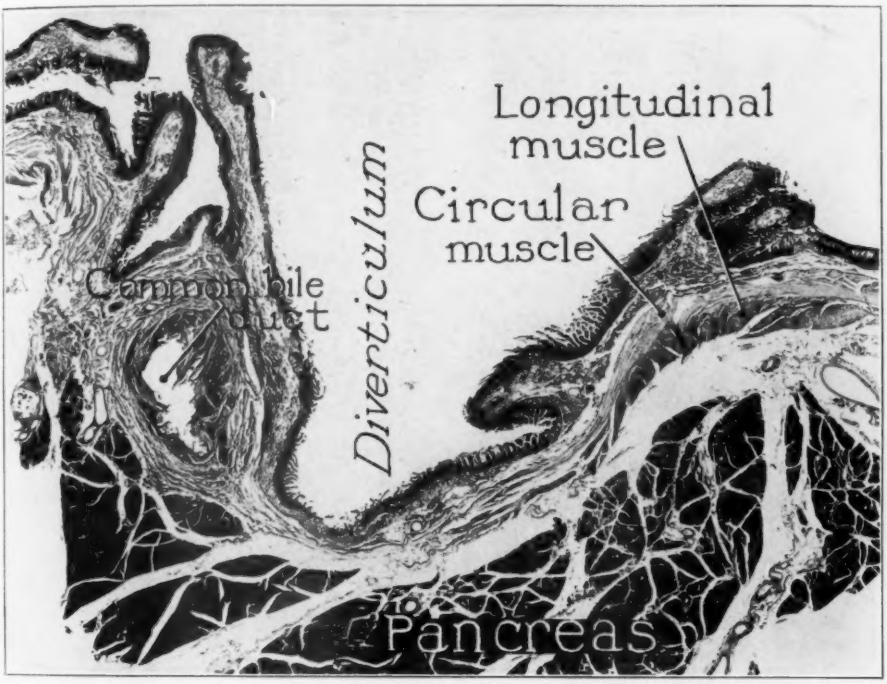

Fig. 2.—Section through diverticulum shown in figure 1 *B*. Strands of muscle tissue almost completely surround the fundus of the sac. van Gieson stain; × 5.

glands in varying proportion were found in a good many of the sections studied, and would, no doubt, have been found more frequently had a greater number of sections been cut from each diverticulum. Lymph follicles were occasionally found. However, in the large sacs of older patients, the mucosa was thin and atrophic, and devoid of Brunner's glands and lymph follicles.

Evidences of previous inflammation were not observed, either in the early or in the old sacs, and no dense connective tissue bundles could be made out beneath the mucosa, as were found in some of the diverticula

Fig. 3.—Section through a diverticulum which is burrowing down beside the common bile duct. The muscle layers may be seen to end rather abruptly shortly before the fundus of the sac is reached. This is an early diverticulum from a man, aged 61. van Gieson stain; × 8.

of the sigmoid and rectum studied for comparison, although in diverticula of the large bowel also atrophic mucosa and the usual muscle layers were absent.

The age incidence in this series of cases in general corroborates that given in the literature. The average age of the patients was 60.8 years. Most of the diverticula were found among persons who had reached the fifth decade of life, but 15 of the patients were between 40 and 49 years of age, and a few cases occurred before the patients were 40 years. The following gives the distribution by decades for the 122 cases: from less

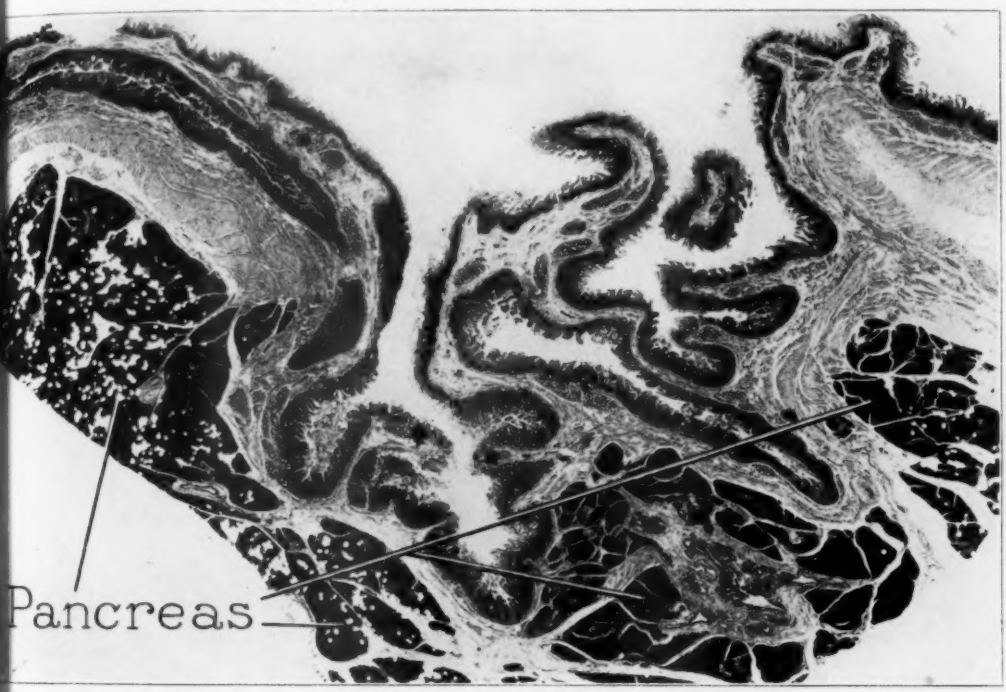

Fig. 4.—Section through an early diverticulum which occurred just proximal to the papilla of Vater in a woman, aged 59. The muscle coats are absent in the fundus of the sac which lies directly on pancreatic tissue, but muscle bundles are present about the plexus of ducts. This diverticulum was probably destined to be bilocular, the ducts resulting in the formation of a septum. The folding of the mucosa, as well as the tissue which contains Brunner's glands and lymph follicles, is unusual, and indicates an early stage in the formation of a diverticulum. van Gieson stain; × 7.

than 1 to 19 years, no cases; from 20 to 29 years, 1 case; from 30 to 39 years, 5 cases; from 40 to 49 years, 15 cases; from 50 to 59 years, 30 cases; from 60 to 69 years, 39 cases; from 70 to 79 years, 26 cases, and from 80 to 89 years, 6 cases. The youngest subject found to have

a duodenal diverticulum was 22 years of age. With the exception of some questionable cases in infants, this is the youngest subject with diverticulum found at necropsy that is recorded in the literature.

An analysis of the ages of the 14 persons who had multiple diverticula disclosed a distribution similar to that of the complete group. The duodenum which was found to have 5 definite diverticula was taken from a woman aged only 41 years.

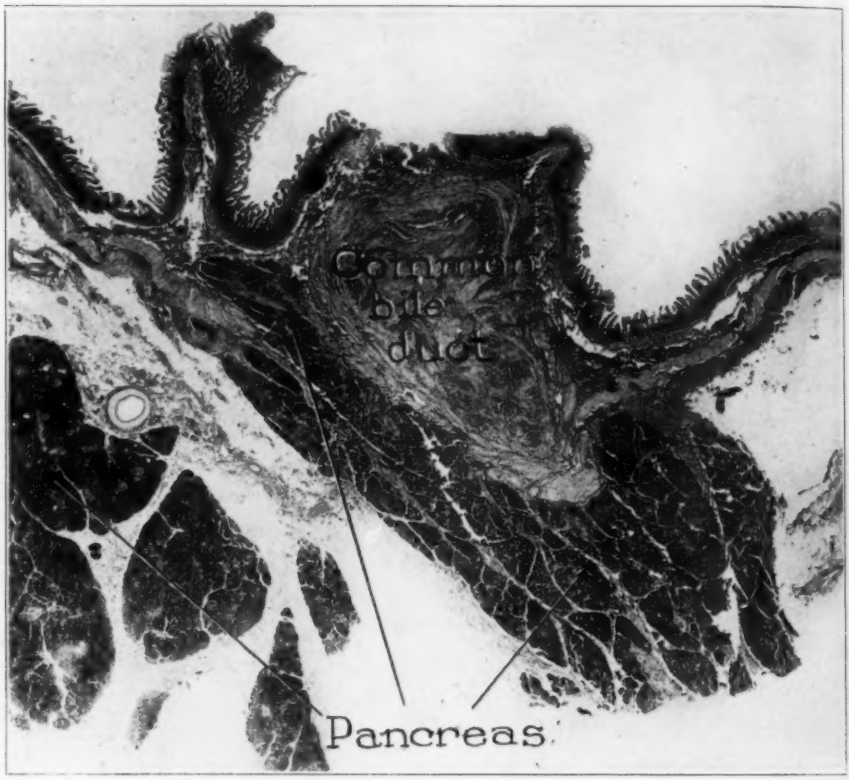

Fig. 5.—Normal duodenum, through the papilla of Vater, from a woman, aged 47. Pancreatic tissue is seen to extend upward to the mucosa, beside the common duct, leaving a small area devoid of muscle, where the mucosa lies immediately over the pancreas. van Gieson stain; × 7.

Of the 122 subjects with duodenal diverticula, 78 were men and 44 women.

In 34 (27.87 per cent) of the cases, there were diverticula of other portions of the gastro-intestinal tract. One was a diverticulum of the esophagus and 1, of the stomach; 2 were Meckel's diverticula; 8, jejunal and ileac diverticula other than Meckel's, and 24 sigmoid and other large intestinal diverticula. In 15 (12.3 per cent) of the cases there were

congenital anomalies, as follows: 2 cases of Meckel's diverticula; 1 case of accessory pancreas (situation not stated), 3 cases of accessory spleens, 1 case of aplasia of the left kidney, 1 case of cystic kidneys, 1 case of congenital cystic pancreas; 1 case of horseshoe kidney, 1 case of malformation of the spigelian lobe of the liver, 1 case of partial duplication of the left ureter, 1 case of a suprarenal rest in the kidney, 1 case of duplication of the gallbladder and 1 case of coarctation of the aorta and duplication of the left renal pelvis and ureter.

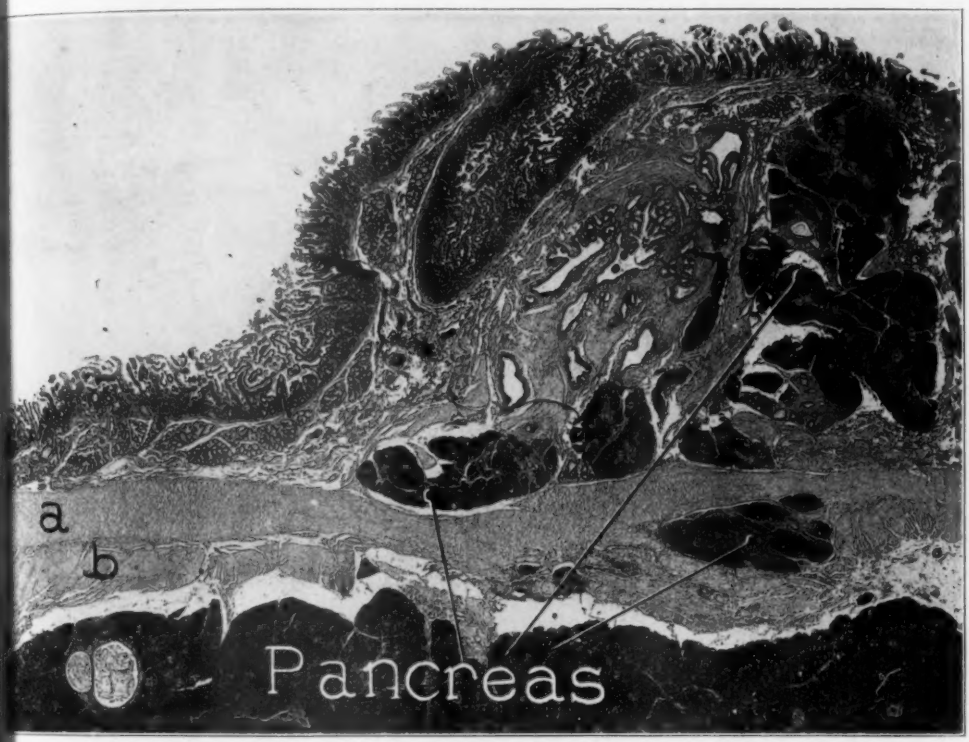

Fig. 6.—Cross-section through minor papilla and duct of Santorini, from a man, aged 47. Between the circular (*a*) and longitudinal (*b*) muscle layers, lie lobules of pancreas, while larger lobules lie beneath the mucosa and about the ducts. van Gieson stain; × 12.

Healed or chronic duodenal ulcer occurred in only 13 cases, and the average distance between the ampulla and pylorus was 7.6 cm. Cholelithiasis or cholecystitis or both were present in 39 cases (31.96 per cent).

In an attempt to find evidences of the precursors of these diverticula, a series of 80 apparently normal and fetal duodenums was studied. Fifty-eight blocks were cut from the region of the minor papilla, seventy-three blocks from the region of the major papilla and fifty-six blocks

from areas from 3 to 6 cm. distal to the major papilla. The entire duodenal wall and the underlying pancreas and connective tissue were included in all these blocks. The tissues were allowed to become fixed in a modified Kaiserling's solution before being cut, in order that the duodenal wall might better retain its relationship to the underlying tissues. The majority of sections were cut at right angles to the long axis of the duodenum, but some were cut parallel to the long axis. Occasional sections were stained with hematoxylin and eosin, but the majority were stained with van Gieson's stain, which gave clear distinction of muscle from connective tissue. Three series of serial sections were cut from the papilla of Vater and the adjacent duodenal wall and pancreas.

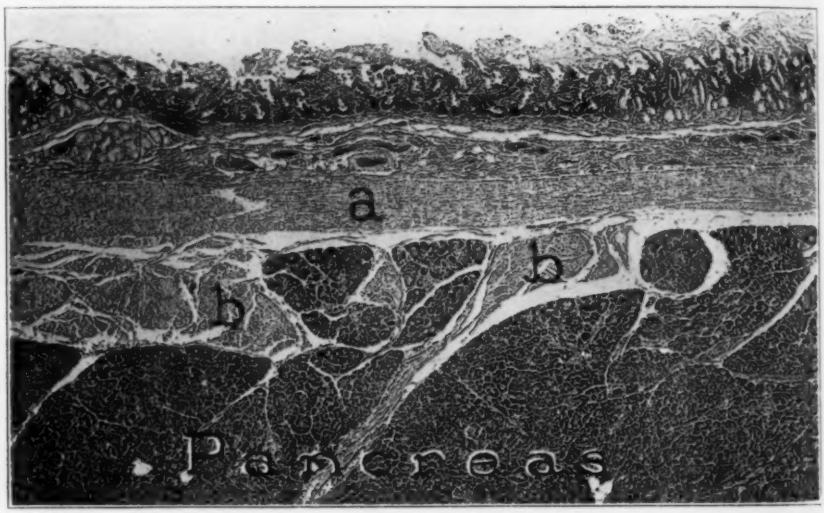

Fig. 7.—Section taken proximal to the papilla in a normal duodenum from a man, aged 37. In *a*, the circular muscle layer is intact, but in *b* pancreatic lobules have broken up the longitudinal muscles; × 25.

Because of the constant relationship of duodenal diverticula to the pancreas, and because of the fact that the duodenal musculature is absent from the diverticular walls, the sections were studied with special reference to the muscle layers and the underlying pancreatic tissue. In most of the blocks cut from the region of the ampulla (66 per cent), it was found that beneath the intramural portion of the common bile duct, just before it opens into the duodenum, the longitudinal layer was absent for a varying distance, although the circular muscle layer was usually intact. The longitudinal muscle layer, when absent beneath the duct, was usually seen to end by penetrating the subjacent pancreas. In the interval between the two ends of longitudinal muscle on either side,

the pancreas lay immediately beneath the circular muscle layer. Sections taken immediately distal to the tip of the papilla, in which case the ducts were not included in the sections, also frequently disclosed that the longitudinal muscle layer was absent for a short distance. However, in those sections taken more than 0.8 cm. distal to the tip of the papilla, both muscle layers were always found to be intact. Blocks which were

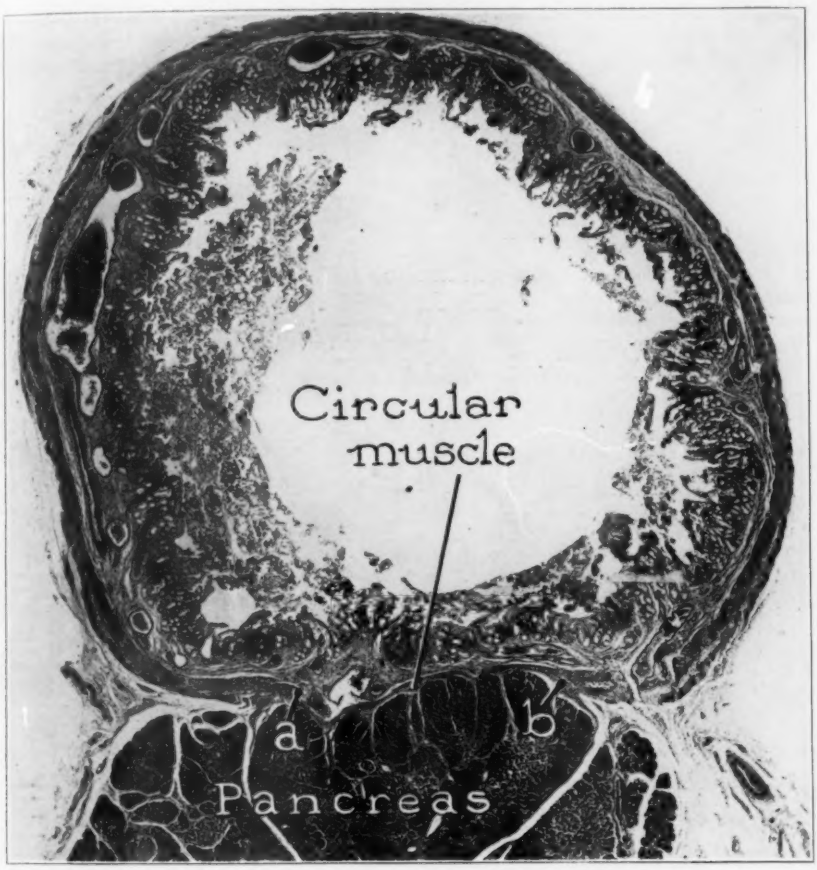

Fig. 8.—Cross-section through the duodenum of a fetus at full term. The circular muscle layer is intact, but at *a* and *b* the longitudinal muscle ends quite abruptly; × 15.

cut just at the point where the common bile duct penetrated the musculature revealed that both layers of muscle were absent where the duct lay, as was to be expected. In these sections it was frequently noted, however, that the longitudinal layer penetrated the pancreatic lobules a considerable distance before the circular muscle ended, either by an abrupt juncture with the musculature surrounding the ducts or by

also penetrating the pancreatic tissue. In a few instances a small lobule of pancreas was seen, lying between the ends of the two muscle layers and the musculature of the duct, immediately beneath the mucosa (fig. 5). In two cases there was pancreatic tissue about the duct beneath the mucosa. The finding of pancreatic tissue beneath the mucosa at the duct of Santorini is of common occurrence (fig. 6), but is seldom seen at the ampulla of Vater.

In a study of serial sections cut through the ampulla and the adjacent duodenal wall, the findings were similar to those just described. The

Summary of Results of Study of One Hundred and Twenty-Two Cases of Duodenal Diverticulosis

		Number	Per Cent
Cases of duodenal diverticula		122	
Cases of multiple duodenal diverticula		14	11.47
Total number of duodenal diverticula		145	
Number associated with other diverticula of the gastro-intestinal tract		34	27.86
Position with relation to ampulla, ascertainable		141	
At ampulla		82	58.15
Above ampulla		36	25.53
Below ampulla		23	16.31
Size:			
Diameter of orifice (average)	1.16 cm.		
Largest	5.00 cm.		
Smallest	0.20 cm.		
Depth of sac (average)	2.03 cm.		
Largest	7.00 cm.		
Smallest	0.30 cm.		
Incidence by age:			
Average	60.8 years		
Oldest	87.0 years		
Youngest	22.0 years		
Sex:			
Men		78	64
Women		44	36
Associated with congenital anomalies		15	12.29
Associated with cholelithiasis or cholecystitis		39	31.96
Associated with duodenal ulcer		13	10.65

circular muscle layer closed beneath the ducts, after they had penetrated the musculature, sooner than did the longitudinal layer, so that in part of the series only circular muscle was present beneath the ducts. To one side of the ampulla, the longitudinal muscle dipped into the pancreas before the ducts were reached, leaving only a thin band of circular muscle between the mucosa and the pancreatic lobules. The mucosa dipped down at this point, and lay close to the muscle, forming the rather deep depression which could be so commonly observed beside the ampulla. In sections cut through the tip of the ampulla, the circular muscle layer was markedly thickened in some areas close to the ducts.

In the region of the minor papilla, or ducts of Santorini, the longitudinal muscle was more frequently (82 per cent) displaced by pancreatic lobules, even in areas some distance away from the ducts (figs.

6 and 7). As has been mentioned, pancreatic lobules were frequently found lying about the plexus of ducts, beneath the mucosa, and in several instances masses of pancreatic tissue lay between or within the muscle layers (fig. 6).

The longitudinal and circular muscle layers were always found to be intact in those blocks cut varying distances distal to the papilla of Vater, except when the blocks were taken within 8 mm. of the papilla, in which case, as mentioned previously, the longitudinal layer was sometimes absent. In a few instances the muscle layers in the lower portions of the duodenum were definitely thinned and atrophic in appearance,

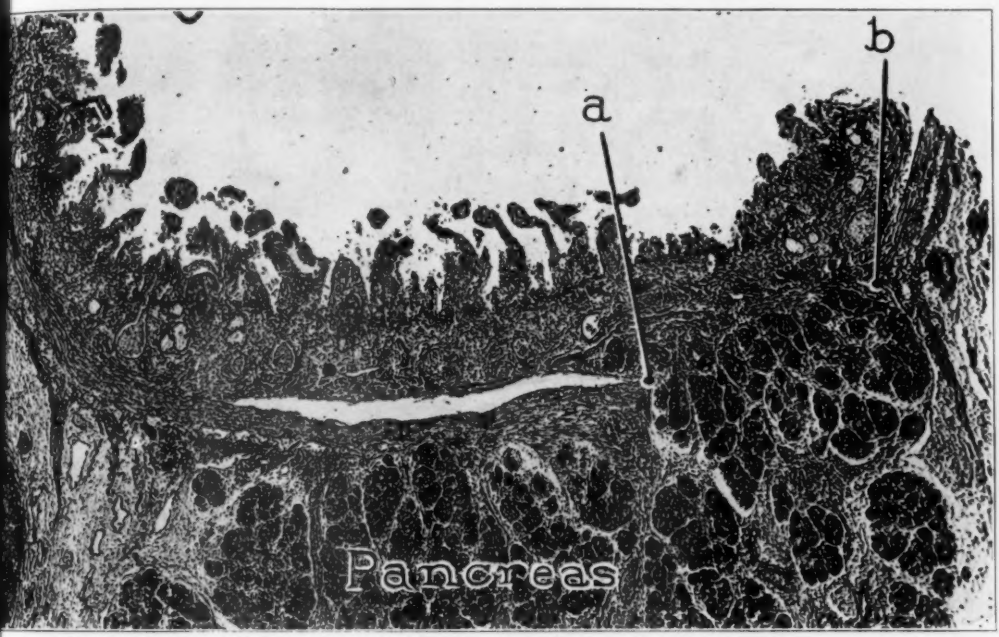

Fig. 9.—Cross-section through the duodenal wall and subjacent pancreas, from a fetus of 5 months. Between *a* and *b* pancreatic lobules have displaced both circular and longitudinal layers. On the left, the longitudinal muscle is seen to dip down into the pancreas and connective tissue. van Gieson stain; reduced from × 45.

and in 4 cases some of the longitudinal muscle bundles were found to extend into the subjacent connective tissue and pancreas.

Sections from the duodenums of 10 fetuses, varying from 5 to 8 months in prenatal development, were studied. Variations in the musculature were found, similar to those just described in the adult. In five of eight blocks cut through the intramural portion of the common bile duct after it had penetrated the muscle layers and lay beneath the

mucosa, the longitudinal muscle was absent for a variable distance and was replaced by pancreatic tissue. In one instance there was a marked thickening of the circular muscle layer close to the papilla. Of seven blocks cut from regions proximal to the major papilla, four contained areas in which the longitudinal muscle was absent and replaced by pancreas, which lay immediately beneath the circular muscle layer (fig. 8). At the points where the longitudinal muscle layers were interrupted, the muscle fibers dipped down beneath the pancreatic lobules. In a fetus of 5 months' development, the pancreas was found to have penetrated the circular as well as the longitudinal muscle layer, in an area proximal to the papilla of Vater (fig. 9).

COMMENT

From the results of this study, an estimate of the incidence of duodenal diverticula could be only roughly approximated. As is well known, when one becomes interested in a particular lesion and begins to search diligently for it, it will be found more frequently than when casual observation of the organ in question is made. This point is brought out by the increased frequency with which duodenal diverticula have been found at the Mayo Clinic within the last few years. Thus, in 1913, 1914, 1915 and 1918, only 1 duodenal diverticulum was found each year, while in 1916, 1917, 1919 and 1920, none was recorded. In 1921, 3 cases were recorded; in 1922, 5, etc. The incidence then gradually increased each year with the exception of 1926; in 1930 the maximum of 2.8 per cent among all postmortem examinations was reached. However, up to June 1, 1931, in the course of 216 abdominal examinations, duodenal diverticula were found in 11, or in more than 5 per cent. Manifestly, since the majority of postmortem examinations at the Mayo Clinic are performed on adult bodies, these figures do not give an idea of the occurrence in a cross-section of the population. The same inaccuracy holds when one attempts to determine the incidence from the literature. Of the larger series of cases taken from records of necropsy, such as Linsmayer's series, the majority come from clinics where a large percentage of the examinations are performed on old subjects, and in these the incidence is higher. Reports from roentgenologists are also inaccurate, since very small diverticula cannot be detected by them, and in addition, they see primarily adults, persons with gastro-intestinal complaints. From various sources then, by rough estimation, the incidence among all persons is perhaps less than 1 per cent, although even this figure seems high if compared with Rankin and Brown's [58] estimate, which is less than 1 per cent for diverticulosis of the entire gastro-intestinal tract.

58. Rankin, F. W., and Brown, P. W.: Diverticulitis of the Colon, Surg., Gynec. & Obst. **50**:836 (May) 1930.

Compared with other diverticula of the alimentary tract, duodenal diverticula stand approximately midway between the most common and the least common. Diverticula of the colon, particularly of the sigmoid flexure, are by far the most common; Meckel's diverticula are said to stand second in frequency, while esophageal diverticula are said to be the most frequent of diverticula of the upper part of the gastro-intestinal tract. From our observations, we should estimate that duodenal diverticula are approximately as frequent in occurrence as are Meckel's diverticula, and more frequent in occurrence than esophageal diverticula. In the stomach and pharynx these anomalies are seldom found.

There has been considerable diversity in the terms used to designate duodenal diverticula. They are commonly referred to as "false" diverticula, this term being used in contradistinction to the "true" diverticula, the walls of which contain all the layers of the intestinal wall, as does a Meckel's diverticulum. "False" seems to us a misleading term, since duodenal diverticula are as truly diverticula as are Meckel's diverticula. Duodenal diverticula have also been termed "congenital" to distinguish them from the "acquired" diverticula associated with duodenal ulcer. This is erroneous, for in few of the cases have the diverticula been proved to be congenital. Odgers introduced the term "primary" to describe these diverticula, using "secondary" to describe the diverticula following ulcer. This is an improvement on the older term "false," but still does not seem satisfactory. The French term, *diverticules périvateriéns,* is correct for those diverticula which occur beside the papilla of Vater, but is obviously incorrect when applied to those occurring in other situations. The old descriptive term used by von Rokitansky [59] with reference to most gastro-intestinal diverticula, namely, "mucosal hernias," is the only descriptive term which is entirely correct and which does not imply more than is accepted concerning these structures. It would seem that no descriptive adjective is necessary in describing these pouches, since "diverticulum" is now practically never used with reference to an ulcer crater or other pouching.

Practically all investigators of this subject are agreed that the walls of duodenal diverticula are made up of mucosa, muscularis mucosa and connective tissue, with occasional bundles of smooth muscle. Only a few exceptions to this statement have come to our knowledge in a review of the literature. Baldwin, in 1911, reported a series of cases and stated definitely that in all there was a layer of muscular tissue in the wall. Case and Bonneau [60] said that the smallest diverticula have muscle in

59. von Rokitansky, Carl: A Manual of Pathological Anatomy, London, Sydenham Soc., 1849-1854, vol. 2, p. 48.

60. Bonneau, Raymond: Diverticules du duodénum, Presse méd. **2**:817 (Sept. 26) 1923.

their walls, while those that are larger are devoid of muscle. Our study has shown that the small, early diverticula have the duodenal muscle coats extending for a variable distance down the sides of the diverticulum, but in no instance was a continuous layer of muscle found extending without break of continuity around the fundus of a diverticulum, as Baldwin described, and we could find no reports similar to Baldwin's in the literature. Our findings have corroborated the majority of the cases reported, in that the fundus of the diverticulum is usually devoid of muscle tissue or contains only a few, small, scattered fibers which can be identified by the van Gieson stain. The muscularis mucosa, as stated previously, was intact in all cases, and in a few instances seemed to be hypertrophied.

As stated by most authors, the mucosa is atrophic, smooth and thinned out in the large and obviously old diverticula. However, in those that are seen earlier in their development the mucosa frequently still retains a few folds and Brunner's glands, and an occasional lymph follicle (figs. 2, 3 and 4). In addition to the presence of mucosal folds and Brunner's glands, the early diverticula also have a different contour; they are either broad and shallow or deep and narrow, but they do not have the bottle-shaped outline of those that are older. This relatively small size of the orifice was emphasized by Linsmayer, who expressed the belief that the orifice retains approximately the size of the actual muscular defect, while the sac expands more and more with age and as pressure is exerted on its wall from within.

As to the histogenesis of these structures, the results of the study of normal duodenums can, we believe, be correlated with that of the structure of the diverticula. In human fetuses as well as in adults, we have found that pancreatic lobules frequently have developed in the position in which the muscle layers of the duodenum should normally develop (figs. 5, 6, 7, 8 and 9), and that this anomaly occurs most often in the region of the pancreatic ducts. It is well known that in embryonic life the epithelial elements of the gastro-intestinal tract, as well as of its offshoots, the liver, pancreas, etc., are well developed before the muscle layers of the adult tube are laid down. Lewis [48] stated that the dorsal and ventral pancreas have been found in 3 and 4 mm. embryos, and that the longitudinal muscle of the duodenum is not developed until the 75 mm. stage is reached, whereas Horton found circular but not longitudinal muscle in a 41 mm. embryo. In addition to the facts given, various investigators [51] have found that in some of the lowest vertebrates, pancreatic tissue is incorporated within the duodenal wall. All these facts, together with the fact that duodenal diverticula lie against the pancreas, without a muscular coat, seem to be convincing evidence that these diverticula arise on the basis of a *locus minoris resistentiae*

produced by an anomalous growth of pancreatic tissue in the wall of the duodenum. This anomaly may be a persistence of the condition found in some lower vertebrates.

The sequence of events may thus be somewhat as follows: The pancreas is laid down at an early fetal age, and the duodenal musculature develops later. If some pancreatic lobules have penetrated the mesenchyma in areas which were to have been occupied by the muscle layers, the muscle cells cannot develop in those areas. Gaps are therefore left in the muscle layer or layers. These gaps are small, but as age advances, or as increased pressure is brought to bear on the duodenal wall, or when the tissues have lost some of their elasticity, the mucosa is forced down through the muscle layers where they are thin or absent, and a diverticulum is formed. The gap in the muscle probably enlarges only slightly, and forms the neck of the sac. The mucosa, however, stretches out gradually, to form the bulbous sac which we see in the well developed diverticulum, while the pancreas gradually gives way to the advancing sac. If the sac continues to enlarge, it gradually pushes out beyond the confines of the pancreas, and lies in the areolar tissue in that region, behind the peritoneum.

This explanation would hold for all the duodenal diverticula which we have examined. It explains their occasional occurrence in young persons as well as their greater frequency in older persons, and why they seem invariably to occur on the concave or pancreatic wall of the duodenum. It seems logical that growth of pancreatic tissue in the duodenal wall should be most likely to occur near the points where the pancreatic anlages first make their appearance as buds from the duodenal epithelium, thus explaining why most diverticula occur in the region of the pancreatic ducts. Linsmayer has emphasized these points particularly, and other investigators have mentioned the influence of accessory pancreatic tissue in weakening the wall of the intestine. One additional point in favor of this argument is the occasional occurrence of pancreatic tissue in the walls of ileac diverticula. Diverticula of the stomach, with accessory pancreatic tissue in their walls, have also been reported occasionally. Thus, although duodenal diverticula are not themselves congenital, the conditions which favor their development in later life are laid down in earliest fetal development.

This study has not substantiated the older and frequently quoted theory that diverticula occur at points of penetration of the duodenal wall by blood vessels. The blood supply of the second and third portions of the duodenum comes from the dorsal and ventral arcades formed by anastomosis of the superior and inferior pancreaticoduodenal arteries, which lie along the concave border of the duodenum, resting on the pancreatic head. Straight branches from these arches penetrate

the duodenal wall from 0.5 to 2 cm. from the line of contact between pancreas and duodenum; that is, the larger arteries do not penetrate the musculature in the area of the wall covered by pancreas. In the specimens dissected no large branches were found penetrating the duodenal wall immediately adjacent to the major and minor duodenal papillae. These facts, in addition to the absence of any microscopic evidence to support it, have led us to discredit the older theory based on the contention that penetration of the duodenal wall by blood vessels causes weakening, with subsequent formation of a diverticulum.

Mailer [61] stated, "Acquired diverticula are probably due, in the main, to intrinsic weakening of the muscle coat of the bowel, incidental to advancing age" and, "the vessel points in the bowel wall are probably not important from the etiologic standpoint, but simply determine the site of the diverticulum in relation to the circumference of the bowel wall." He also pointed out that numerous diverticula of the colon do not confine themselves to the points of penetration of vessels, but crop out all over between the mesocolic and anterior longitudinal muscle bands, as a result of generalized weakening of the musculature. It is probable that acquired diverticula of the entire alimentary tract, from esophagus to anus, owe their origin to inadequacies, either developmental or acquired, in the musculature of the wall of the intestine. The greater incidence of diverticula of the large bowel is no doubt closely linked with the fact that the longitudinal muscle layer is limited to three bands, and it is in the part of the wall devoid of longitudinal muscle that the diverticula of the colon usually occur.

If the foregoing explanation as to the mode of formation of duodenal diverticula is correct, sex should be of little significance in their development. We believe that this is true, in spite of the fact that in this series the patients were preponderantly men, with approximately a 2:1 ratio of men to women. Most authors give a greater percentage of diverticula in females, but Buschi found them more frequently in males. These discrepancies between the observations of various workers are probably due to the fact that none of the series of cases is large. Probably, if all cases were pooled, duodenal diverticula would be equally distributed between males and females.

The histories of the cases comprising this series were not reviewed minutely, but it seems improbable that these diverticula, which were in all instances found incidentally at necropsy, should have produced symptoms. None gave gross or microscopic appearance of inflammatory changes, and in all the mucosa was smooth and unbroken. We do not agree with the more recent writers, particularly the roentgenologists,

61. Mailer, Robert: Observations on Diverticula of the Colon: Pathological and Clinical Study, Lancet **2**:51 (July 14) 1928.

that duodenal diverticula are frequently a menace to the health and life of the patient. Occasional cases have been reported of a ruptured diverticulum, or of obstruction due to pressure of a diverticulum, and theoretically this might occur, but we believe that complications due to duodenal diverticula are exceptions rather than the rule. In 31.96 per cent of this series, the diverticula were associated with cholecystitis or cholelithiasis, and the average distance between the ampulla and pylorus was 7.6 cm. Robertson and Hargis gave 7.9 cm. as a normal average distance between ampulla and pylorus. In a series of 100 consecutive postmortem examinations of unrelated cases performed during 1931, cholecystitis or cholelithiasis was found in 24 per cent. The latter figure is lower than the 31.96 per cent of instances of cholecystitis or cholelithiasis given as having been found in the series of cases of duodenal diverticulum, but neither group from which these percentages were derived was large, and it would seem that the two figures could be considered as analogous. In 17 per cent of the same group of 100 postmortem examinations congenital anomalies were found. This figure is to be compared with 12.3, the percentage of congenital anomalies in the series of cases of diverticulum. Here the control group gives a higher percentage. This is probably in large measure due to the fact that more minute scrutiny of all organs has been carried out and all deviations from the normal have been recorded in recent years, whereas in the earlier protocols only the very obvious lesions were recorded. Thus, of the two groups of cases, the control series is more accurate. The percentage for the series of cases of diverticulum is probably considerably lower than it would be, had present-day criteria been employed. These comparisons are therefore made with reservations and are probably of little value.

The surgical hazards which might arise from the presence of a diverticulum should be mentioned. An unidentified pouch might readily be mistaken at operation for a cyst, a dilated duct or some similar structure. The danger of formation of a duodenal fistula, which would jeopardize the patient's life, should therefore be constantly kept before the surgeon's mind.

CONCLUSIONS

1. Duodenal diverticula are hernias of the duodenal mucosa through a defect in the muscular layer, the fundus of the sac usually being devoid of musculature.

2. They occur most frequently in the region of the papilla of Vater and the duct Santorini, but may be found in other portions of the duodenum.

3. All duodenal diverticula in this series were situated on the concave or pancreatic border of the duodenum, with some part of the diverticulum in contact with pancreatic tissue.

4. At necropsy they are most frequently found in subjects who have passed the fifth decade of life, but they may occur in young subjects.

5. The large and obviously old sacs are bottle-shaped, while the early diverticula may be broad and shallow.

6. Duodenal diverticula are not congenital, but probably develop in adult life on the basis of a *locus minoris resistentiae* produced by an anomalous congenital growth of pancreas within the duodenal wall.

7. The wall of the apparently normal duodenum in the fetal and adult state may contain pancreatic lobules which displace muscle fibers, thus producing a *locus minoris resistentiae.*

8. Duodenal diverticula probably seldom cause clinical symptoms or serious complications, but it is possible that an unidentified pouch might form a serious hazard in the course of operation on the upper part of the abdomen.

9. The diverticula in this series were associated with congenital anomalies in 12.3 per cent of cases, and with other diverticula of the alimentary tract in 27.8 per cent.

UNILATERAL SPINE FUSION

A SIMPLIFIED TECHNIC

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The operation of spine fusion is long past the experimental stage and is established as a valuable procedure in structural scoliosis, fracture of the spine, destructive disease of the vertebrae, spondylolisthesis and similar conditions. The operation is advised with reserve by many surgeons because the technic is difficult. In the hands of the experienced spine fusion may be accompanied by shock, which at times borders on being dangerous. It therefore occurred to me that it might be possible to circumvent these difficulties and yet attain the primary object of the operation, namely, fusion of the laminae and spinous processes, by employing a simplified technic. The idea originally grew out of my experience in scoliosis. In the very severe types of this deformity one is compelled to do a unilateral fusion, as it is frequently difficult, and sometimes impossible, to expose thoroughly the posterior arches on the convex side of the curve. In many cases the apparently incomplete operation was entirely satisfactory. Unilateral spine fusion, according to the method about to be described, is especially applicable to lesions of the spine in which there is little or no lateral distortion of the vertebrae, and to the lower half of the spine in which the spinous processes are fairly thick, that is, from the tenth dorsal to the sacrum.

TECHNIC

Let it be assumed that one is dealing with a case of tuberculosis of the first lumbar vertebra. A vertical incision is made in the median line of the back from the tenth dorsal to the third lumbar spinous process. The incision is extended through the superficial and deep fascia, immediately bringing into view the spinous processes (fig. 1). With a sharp thin chisel the eleventh dorsal spinous process is split in an anteroposterior direction; the left half is left attached to the vertebra, while the right half is split off from the lamina near its base. A periosteal elevator is inserted under the split off half of the process, and the process and the periosteum on the right side of the arch are elevated from the underlying bone to the articulation. The interspinous ligament is cut through in an anteroposterior direction, and the right half is retracted outward with a periosteal elevator. The same procedure is carried out on the other spinous processes and the interspinous ligaments. Within a few minutes and with little trauma the laminae on the right side of the

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This method was presented at a meeting of the Clinical Society of the Hospital for Joint Diseases, New York, Jan. 5, 1932.

vertebrae are exposed. The retracted mass of tissue contains muscle, five large segments of bone, the corresponding interspinous ligaments and the periosteum of the laminae (fig. 2). Under this thick layer of tissue a graft of beef bone is placed on the laminae near the articulations (fig. 3). Frequently, before placement of the graft, chips of bone are removed from the laminae and placed across the interlaminar spaces. The sheet of muscle, bone and periosteum is then mobilized by two incisions through it, one at each extremity. With gentle traction the split portions of the spinous processes are brought into the interspinous areas and into contact with the unsplit segments (fig. 4). They are held in place with interrupted strong

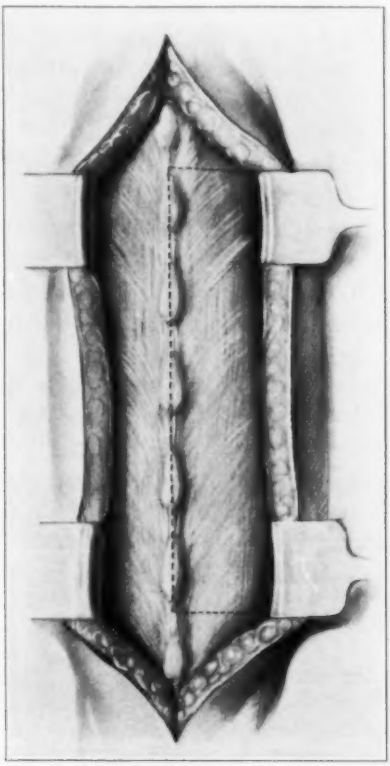

Fig. 1.—The vertical dotted line indicates the incision through the spinous processes and interspinous ligaments. The horizontal dotted lines indicate the incision through the periosteum to allow mobilization of the sheet of tissue containing the split off portions of the spinous processes, the periosteum, the interspinous ligaments and the muscles.

number 3 or 4 chromicized catgut sutures passed through the strong fibroperiosteal covering on the spinous processes. The wound is closed with a layer of catgut sutures for the deep fascia and another for the superficial fascia and a layer of silk sutures for the skin.

As the steps of the operation are few and the technic is simple, the actual operating time is rarely more than from fifteen to twenty

minutes. Consequently the patient has only slight postoperative depression and within twenty-four hours feels entirely comfortable. Compared with ordinary spine fusion, this operation causes very little pain.

The essential features of the type of spine fusion here described are: 1. The spinous processes in the area operated on are bridged over by masses of their own bone, and hence they readily fuse and form a long, strong, bony bar supporting the affected part of the spine. 2. The

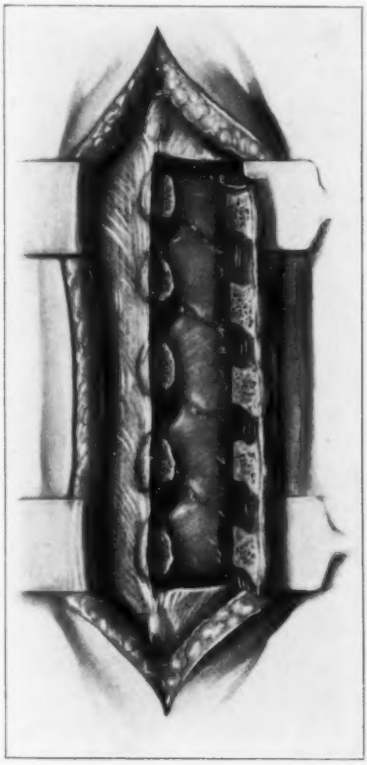

Fig. 2.—Drawing showing retraction of the tissues elevated from the right side of the posterior arches, including the split off portions of the spinous processes.

beef bone graft held in intimate contact with the laminae by the layer of muscle and periosteum gives additional support to the spine. In time it becomes replaced by new bone which unites the laminae.

The specific advantages of the operation described are:

1. The operation can be performed in a short time.

2. There is a minimum amount of manipulation and trauma.

3. The existing support of the spine is not disturbed, as half of each spinous process is left intact.

4. The patients have much less discomfort than from the more extensive operation of fusion.

5. Postoperative shock is eliminated by the short anesthesia and the simple technic.

6. Fusion occurs unusually fast because the spinous processes are brought into contact with segments of their own substance.

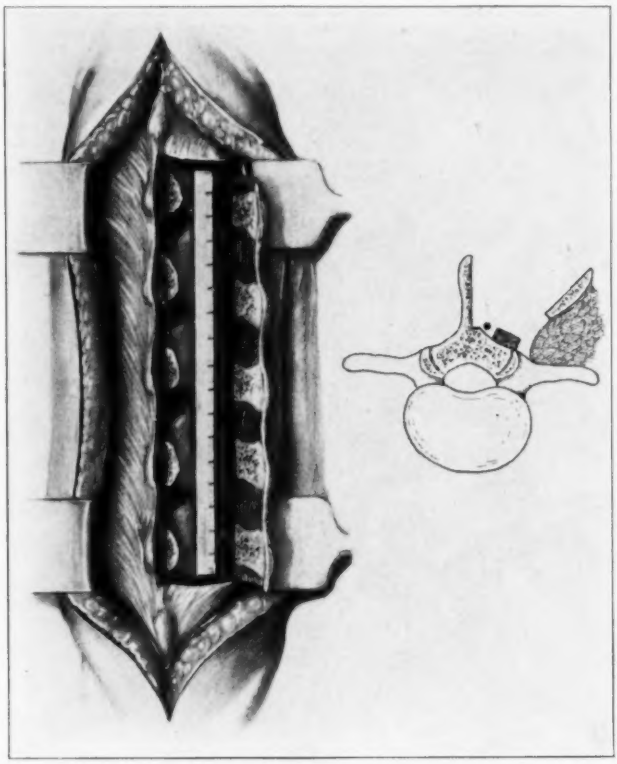

Fig. 3.—The same view as that shown in figure 2. A beef bone graft has been placed on the laminae covering the articulations. The small insert is a diagrammatic sketch showing half of the spinous processes split off and retracted outward with the periosteum and muscles. The small dark square represents a cross-section of the bone graft placed on the lamina near the intervertebral joint.

7. The ultimate support of the affected area of the spine comes from fusion both of the spinous processes and of the laminae.

As the spinous processes of the upper and middorsal vertebrae are comparatively thin, the operation of unilateral spine fusion is limited for the present to lesions in the lower half of the vertebral column, that is, from the ninth or tenth dorsal vertebra to the sacrum. In the

lower half of the spine the spinous processes are long and thick and permit the splitting off of large fragments without disturbance of the support of the back. So far this operation has not been used in structural scoliosis, in which it is difficult to be certain of the shape, size and inclination of the spinous processes without thoroughly exposing them. The operation is especially applicable to fractures or dislocations of the spine and to a destructive lesion without lateral deformity.

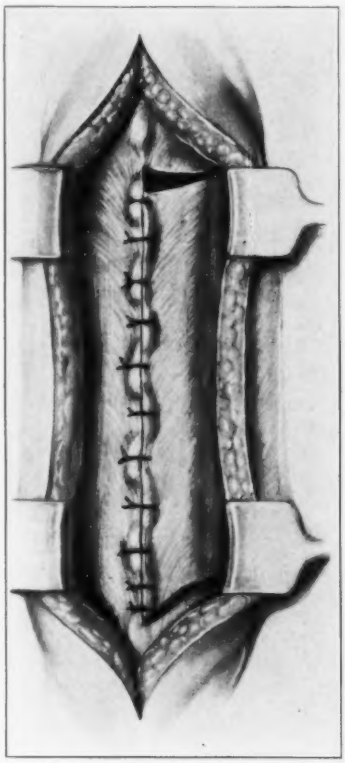

Fig. 4.—Drawing showing mobilization and downward displacement of the mass of bone, periosteum and muscle. The split off portion of the spinous process of the uppermost vertebra has been moved down and brought into contact with the lower extremity of its own spinous process and with the upper part of the spinous process below. The same procedure is carried out with every fragment, with the result that there is a continuous mass of bone along the line of the spinous processes.

REPORT OF CASES

CASE 1.—George Le To, aged 40, had a fracture of the second lumbar vertebra for which it was decided to do a spine fusion. He was operated on at the Hospital for Ruptured and Crippled on Sept. 25, 1930. The twelfth dorsal to the fourth lumbar vertebrae, inclusive, were operated on. The technic employed was that

already described. The total time of the operation was twenty-one and a half minutes, at the end of which time the patient was in excellent condition. The postoperative course was uneventful. Within two days the patient was entirely comfortable. He was discharged from the hospital at the end of seven weeks. A spinal brace was applied three months after the operation and discarded six months later, when support was no longer necessary. At the time of this report, one year after operation, the patient has no backache and can lift 100 pounds (45.4 Kg.) without much effort. In the roentgenographic lateral view of a lumbar spine that has not been operated on the spinous processes are separated by large distinct

Fig. 5.—Lateral view of a normal lumbar spine. Note the clear interspinous spaces.

intervals (fig. 5). In the postoperative lateral view of this patient (fig. 6) one sees the darker shadows of the undisturbed portions (*a*) of the spinous processes, and between them are lighter shadows (*b*) of the displaced segments of bone bridging the interspinous areas. The body of the fractured vertebra is connected to the adjacent vertebral bodies by large bridges of bone. Thus the area of fracture is well protected by bony union of the injured vertebra to those above and below it both anteriorly and posteriorly.

CASE 2.—Joseph L., aged 55, had a fracture of the first lumbar vertebra. He was admitted to the Hospital for Ruptured and Crippled and was operated on on

Nov. 11, 1930. The eleventh dorsal to the third lumbar vertebrae, inclusive, were operated on according to the technic described. The time of operation was twenty-four minutes. On the following day the patient was quite comfortable. At the end of seven weeks he was discharged from the hospital. A brace was applied two months later, and physical therapy was instituted. He has made satisfactory progress and has obtained fusion of the operated area. At the time of this report, one year after operation, he is going about without any support and has no pain in his back.

Fig. 6 (case 1).—Lateral postoperative view showing compression fracture of the second lumbar vertebra. The letters, *a, a, a,* indicate the undisturbed portions of the spinous processes of the first, second and third lumbar vertebrae. The split off portions of the spinous processes are seen as lighter shadows in *b, b,* bridging the interspinous areas.

Case 3.—Mrs. Frances S., 36 years old, in a fall down a flight of steps sustained a fracture of the first and a dislocation of the fifth lumbar vertebrae. She was operated on at the Hospital for Joint Diseases on March 20, 1931. Since two areas needed fusion, it was important that the operation be so planned as not to be excessively long. The technic described in this paper was of enormous advantage. An incision was made over the twelfth dorsal and first and second lumbar vertebrae, and the operation was carried out within about fifteen minutes. A

similar operation was carried out lower down, including the fourth and fifth lumbar and the first and second sacral segments. The procedure was performed thoroughly, and yet the total time required for both operations was less than an hour. This patient, too, has done well. She is now walking about freely, wearing a spinal brace, and has no discomfort in her back.

Case 4.—Miss K. R. was a patient of my associate, Dr. Buchman, who gave me the details in this case. For two years she had been suffering from tuberculous disease of the upper lumbar vertebrae. Psoas abscesses had developed. She had high fever for many weeks and was in poor general condition. The abscesses had to be drained. The patient was kept in bed on a convex frame for several months. Finally she improved sufficiently to warrant the undertaking of an operation. It was manifestly important to obtain fusion through an operation that would be thorough and yet of short duration and involve a minimum of trauma. The tenth dorsal to the fifth lumbar vertebrae, inclusive, were operated on. The technic described in this paper was used. The patient stood the operation well, and is convalescing satisfactorily.

SUMMARY

A simplified technic for spine fusion is described. Its chief advantages are simplicity and ease of execution.

The procedure of unilateral spine fusion permits thorough preparation of the vertebrae for fusion without removal of the bony support of the spinous processes. Union of the spinous processes is assured by bridging them with segments of their own substance. The time of operation is relatively short, and the manipulation is gentle. Hence this operation may be employed in patients of advanced age, and also in those who are debilitated and too sick to be subjected to prolonged anesthesia and severe operative procedures.

Additional Cases.—During the six weeks preceding the writing of this report I used this technic in several additional cases in my service at the Hospital for Joint Diseases. One patient was a child of 7 years with tuberculous disease of the seventh dorsal vertebra. The fifth to the ninth dorsal vertebrae, inclusive, were operated on. Contrary to my expectations, I was able to employ the technic described. The only difficulty was the need for caution in splitting the spinous processes, which were very thin. The second case was in a girl of 15 years with moderate structural scoliosis. Fourteen vertebrae were operated on. The technic was carried out thoroughly, and the time of operation was fifty-six minutes. The patient was in good condition after the operation. During the last week I operated on a third patient, an adult suffering from tuberculosis of the third and fourth lumbar vertebrae. All of the lumbar vertebrae and the first sacral segment were fused. The operation, without any attempt at speed, was completed in a little more than thirty minutes.

PLASTIC OPERATIONS FOR INCONTINENCE OF URINE AND OF FECES

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Incontinence of urine and feces are conditions so distressing to patients and offensive to their associates that it is not surprising that every conceivable method has been employed in attempts to relieve them. In that group of cases in which the incontinence is due simply to the sphincter having been cut across, accurate approximation of the divided ends usually results in complete restoration of function. Even when it has been divided in two or more places, or partially destroyed, operations which restore to use such muscle as remains, completing the ring with fibrous tissue of some sort, are often successful. But when the sphincter has been severely damaged, has been removed altogether, is congenitally absent or has had its nerve supply destroyed, the problem becomes far more difficult, and the prognosis correspondingly poorer. The multiplicity of operations devised only serves to show how earnest but vain has been the search for a satisfactory solution.

In 1927, Wreden of Leningrad attacked the problem of anal incontinence in a new way. He employed the contracting force of the gluteal muscles left in situ, transmitting this pull to the anus by means of strips of fascia, which act passively, much as a tendon does. He thus avoided the faults inherent in closed fibrous rings, and in transplanted strips of muscle, which sooner or later lose the power of contraction. He reported [1] one successful case in which he placed two loops of fascia lata subcutaneously, like interlocking links of a chain, the interlocking portions enclosing the anal orifice, and the lateral portions clasping a bundle of gluteal muscle fibers on either side. The pull of these two loops against each other produces the sphincter action. Dr. Harvey Stone [2] of Baltimore, using a slightly different technic, and prepared fascia instead of an autotransplant, has reported two cases, one of which was successful. Ransohoff [3] has also reported a successful case.

From the Department of Surgery, Shantung Christian University Medical School and Hospital, Tsinan, Shantung, China.

1. Wreden, R. R.: A Method of Reconstructing a Voluntary Sphincter Ani, Arch. Surg. **18**:841 (March) 1929.

2. Stone, H. B.: Plastic Operation for Anal Incontinence, Arch. Surg. **18**: 845 (March) 1929.

3. Ransohoff, J. L.: Wreden's Method of Reconstructing Voluntary Anal Control, Ann. Surg. **90**:317, 1929.

A Chinese girl, with an unusual combination of congenital abnormalities, presented the double problem of incontinence of feces and urine, due to defective innervation. The operation of Wreden seemed particularly well adapted to the former, and accordingly was carried out.

REPORT OF A CASE

History.—Ma Mao, a Chinese girl, was first seen in the outpatient department in 1924. She was then 10 years old. The diagnosis at that time was: transposition of viscera and incontinence of urine and feces since birth. Admission to the hospital was advised, but since cure could not be guaranteed, she was taken away. During the following years large quantities of Chinese drugs were consumed, and various "old-time" treatments were endured in the effort to gain relief.

Examination.—On Nov. 26, 1930, she was brought again to the outpatient department. She was now 16 years of age, but looked not more than 13. Her height was 4 feet and 4 inches (132.1 cm.) and she was rather heavy-set. She appeared well nourished and in good health. Examination showed a true transposition of both thoracic and abdominal viscera. The breasts were small; the axillary and pubic hair was scanty. The clothing was wet with urine and soiled with feces. The skin of the perineum, buttocks and inner aspects of the thighs was excoriated and painful. The gluteal muscles were poorly developed. The anus was deeply depressed, and above and behind it, in the space normally occupied by the sacrum and coccyx there was a large hollow. The labia minora were elongated. The urethral meatus was in the normal position. Urine dribbled out constantly during examination. No vestige of the hymen could be seen. The vagina was surprisingly large and deep, but otherwise normal in appearance. In knee-chest posture the anal sphincter gaped widely, and then closed slowly and feebly. Digital examination showed a thin, weak, diaphragm-like external sphincter; the internal sphincter could not be made out. The rectal wall looked and felt normal.

The following day the patient was admitted to the University Hospital for study and treatment.

Routine work-up brought out some additional facts. The past history revealed that the patient had been in excellent general health; she had had measles in early childhood, had never had chronic constipation and for a year had menstruated regularly every four weeks without unusual discomfort. Physical examination showed one carious molar and pyorrhea; the heart (except for transposition) and lungs were normal. The tubes and left ovary were not definitely felt. The bladder (inspected through an open-air Kelly cystoscope) appeared normal, except that no sphincter action could be demonstrated on removal of the instrument. The legs were rather small, but strong. A chilblain ulcer was noted on the left foot. Systematic neurologic examination gave negative results except for unusually active knee reflexes. Laboratory examination of the urine revealed cloudiness, a trace of albumin; red blood cells, white blood cells and many bladder epithelial cells. Examination of the feces showed occult blood and many Ascaris eggs. There were 4,102,000 red blood cells, 80 per cent hemoglobin, 7,400 white blood cells and the differential count was normal. The Wassermann reaction was negative. Renal function (phenolsulphonphthalein intravenously) was 10 per cent the first, and 27 per cent the second, hour. Roentgenographic examination was made (figs. 1 to 7).

The diagnosis was: complete transposition of viscera; congenital absence of the coccyx and sacrum; congenital incontinence of urine and feces due to defective innervation; slight underdevelopment, general, sexual and mental; chronic low grade infection of the urinary tract; ascariasis; oral sepsis, and a chilblain ulcer on the foot.

The patient was kept under observation for four weeks. During this time her general condition improved markedly; she was rid of her intestinal parasites; the urine became normal; renal function rose to 86 per cent in two hours; the skin of the perineum and buttocks improved daily with nursing care and the liberal use of ointments; the oral sepsis and ulcer on the foot received attention, and there was

Fig. 1.—Roentgenogram of the chest, showing a true dextrocardia and gas in the stomach on the right. There are twelve thoracic vertebrae.

gain in weight. There was no appreciable improvement in the ability to control the urine or feces.

Operations and Course.—Wreden's operation was carried out on December 23 under ether anesthesia. The gluteal muscles were misshapen and small, probably due to the absence of sacrum and coccyx, so it was necessary to make the lateral incisions somewhat farther lateral and posterior than otherwise would have been necessary. When uncovered, the muscles appeared to be composed largely of fibrous tissue. Two strips of fascia, 19 cm. long, and 1 cm. wide, were cut from the right thigh. The remainder of the operation followed closely the technic suggested by Stone. It was sheer guess-work as to how tight to draw the interlocking loops. The operation itself was surprisingly easy. The anus assumed the usual puckered appearance, and through the anal walls the fascial loops felt much like a normal sphincter.

Fig. 2.—Roentgenogram showing the absence of the sacrum and coccyx. There are four lumbar vertebrae. The sacrum is represented by one large, irregularly-shaped vertebra.

Fig. 3.—Lateral view of the pelvis, showing absence of the sacrum and coccyx.

Fig. 4.—Roentgenogram taken after a barium meal, showing the fundus of the stomach on the right and the pylorus on the left.

Fig. 5.—Roentgenogram taken twenty-seven and a half hours after a barium meal. The barium is found in the cecum and ascending colon, which are situated near the midline, and in the transverse colon, which is shown running up to the splenic flexure in the right hypochondrium. A small amount of barium had been expelled from the rectum when this exposure was made.

Fig. 6.—Roentgenogram taken after the injection of 800 cc. of barium emulsion into the rectum. The hepatic flexure is clearly shown. The shadow at the bottom is a barium-soaked cotton pad pressed against the incontinent anus.

Fig. 7.—Roentgenogram taken after an attempt to expel the enema.

Because of the constant leakage of urine, the wounds were covered with evaporated compound tincture of benzoin, and the patient was kept prone for five days. The incisions healed without the least inflammation. In the light of Stone's first experience, my colleagues and I decided to take advantage of this period and teach the patient to use her gluteals to keep her new "sphincter" closed. We failed in this.

On the eleventh day she was given liquid petrolatum and passed three controlled stools, greatly to her own surprise. Thereafter she had complete control of solid and semisolid feces. After vigorous catharsis, deliberately tried, there was some involuntary leakage of liquid feces. Figure 8 shows the record of defecations during three of the four weeks under observation. As will be seen, there was some tendency to constipation. Except for the incident just referred to, there was no leakage of feces during this period.

Encouraged by the apparent success of this operation, we sought by some similar means to relieve the incontinence of urine. It soon became evident, however, that the principle used in the first operation was not applicable to the urethra. Not only is the urethra so situated that interlocking loops of facia cannot very well be placed about it, but there are no available muscles on either

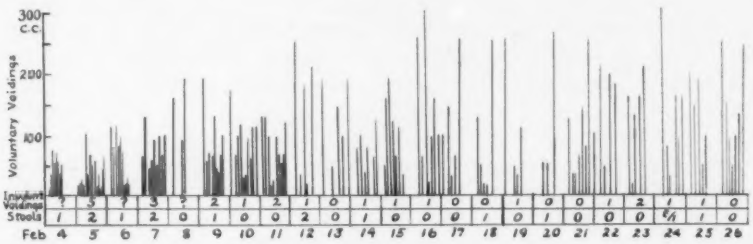

Fig 8.—Chart showing the number of controlled stools, the number of times urine was lost involuntarily, and the number and amounts of voluntary voidings, from the third to sixth postoperative week. A few voluntary voidings were thrown out, or were not measured separately, and so cannot be included. It will be noted that the largest amounts, as a rule, were passed the first thing in the morning.

side to which the lateral ends of the loops might be attached, except the muscles of the thighs, and here the movements of the legs would surely defeat the purpose of the operation. It occurred to us finally that voluntary constriction and relaxation might be obtained by fixing the distal end of a loop of fascia to a movable part of the body, so that the pull would be exerted, not by the contraction or elasticity of tissues so much as by the voluntary assumption of postures. For this purpose it was necessary to find some nearby portion of the body that in ordinary positions, such as standing and sitting, would be at a greater distance from the urethra, and in some extraordinary position, such as squatting or extreme flexion of the spine, would be nearer, and so relax the habitual tension on the fascial loop. After some experimenting, it was found that the rectus muscle a short distance above the symphysis most nearly fulfilled these conditions. The muscles were chosen rather than their fascia because of the inelasticity of the latter, which might subject the urethra to sudden, and possibly violent, pulls. It was decided, therefore, to attempt to sling a strip of fascia under the urethra near the bladder, with the two arms passing between the bladder and symphysis upward to be attached to the rectus muscle on either side.

Before operation a careful cystoscopy was done. The bladder mucosa was found to be normal everywhere. The urethral orifices appeared normal, and urine could be seen coming from them in normal spurts. A sense of fulness was produced by distending the bladder with 100 cc. of fluid, and this fluid could be expressed by the patient with a force exceeding the weight of 30 cc. of water. We concluded that in size, sensation and muscular power the bladder was potentially normal.

Operation was performed on Jan. 16, 1931, under ether anesthesia. One hundred cubic centimeters of air was injected into the bladder, the rubber catheter being clamped and left in place. The patient was placed in Edebohl's lithotomy position. A strip of fascia as long as could be obtained (19.5 cm.) and 2 cm. wide was taken from the the left thigh. An 8 cm. incision was then made above the symphysis pubis, the recti exposed on either side, and freed from their fascia medially. The peritoneum was rubbed off the anterior surface of the bladder out of harm's way. It was relatively easy to push the bladder away from the symphysis and expose the roof of the urethra; but when we attempted to pass an instrument under the urethra and up on the other side, it proved surprisingly difficult. None of our instruments seemed to be the right size or shape. First I tried, with one hand in the wound and the other under the sheets in the vagina, to direct the point of an aneurysm needle between the urethral and vaginal walls at a point about half way between the urethral orifice and neck of the bladder, an assistant manipulating the handle. Between us the instrument slipped somewhat out of place, and the bladder was perforated near the midline posteriorly, with escape of air and urine. The bladder had to be subjected to a wide dissection in order to close this hole satisfactorily. With this improved exposure, working from above, I was finally able to pass the aneurysm needle under the urethra, a little nearer the distal end of the canal than I had originally planned. A nurse with a finger in the vagina helped to prevent perforation of its mucosa. A piece of strong catgut was pulled through this tract, and in turn the strip of fascia (fig. 9). Because the fascial strip was not as long as we could have wished, the end of each arm was split into two fingers, and these were fastened around a generous portion of rectus muscle on either side, 5 cm. above the upper border of the symphysis. It was then found that the bladder tended to herniate between the easily spread arms of the V; otherwise the suspended fascia would prevent the anterior wall of the bladder from approaching the inner surface of the pubes, and thus create a dead space. Therefore, we sewed the muscles together behind the transplanted fascia (fig. 10). The ends of the sling were drawn up and fastened tight enough to prevent the rubber catheter from being withdrawn readily. The upper portion of the sling lay between the recti, and did not come in contact with the sheath, which was closed anterior to it. A small celluloid-tissue drain was put in the lower angle of the wound.

The catheter was left in the bladder, but it did not drain well; the following day the end of it was found in the vagina. So the patient was put in Fowler's position with the knees well flexed. In this position there was free discharge of urine, and we were dubious about the success of the operation. There was low grade fever for a week. The suprapubic wound became infected, and the discharge showed a pure culture of B. coli. Two-hourly irrigations of the wound with saturated boric acid solution through a Carrel tube brought about rapid improvement, the bacterial count dropping from 18 to zero in a few days.

The patient was allowed to walk on the fifteenth day, and the nursing staff undertook to train her to control her urine. For two or three days she was kept in the toilet, and urged to respond immediately to every slight impulse to urinate.

As soon as she learned that it was possible for her to go a whole day without wetting herself, she became interested in the training and made rapid progress. She learned the difference between sitting with and without flexing the spine. Figure 8 shows the improvement in control during the first three weeks of this training. The largest single amount voided was 350 cc. on the day of her discharge, March 6, forty-six days after operation. More than once she was able to go through a whole night without voiding. But her control was not perfect at the

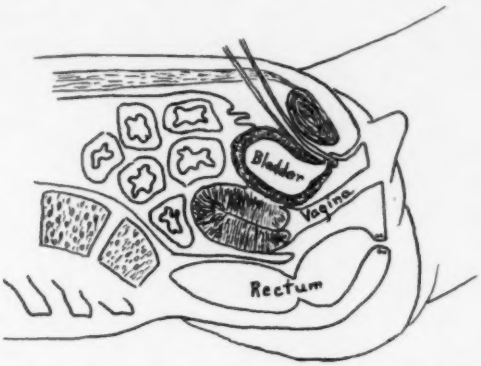

Fig. 9.—Schematic drawing of the pelvis in longitudinal section, showing the fascial sling placed about the neck of the bladder, and between the symphysis and anterior wall of the bladder.

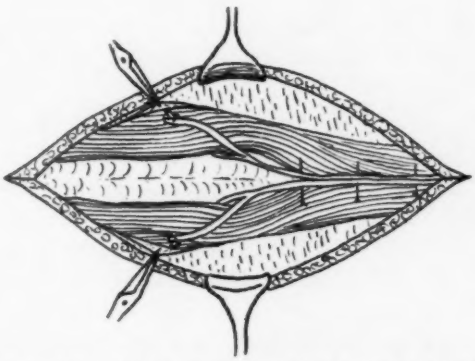

Fig. 10.—Showing the ends of the strip of fascia attached to the rectus muscle on either side, and the approximation of the muscles behind the fascial sling.

time of her discharge. Accidents still occurred every day or two, sometimes at night, sometimes because she indulged in the habit of squatting on her haunches to rest herself, and once or twice because of emotional upsets.

Examination on the day of discharge showed all the wounds thoroughly healed. The new anal "sphincter" felt much like a normal tight sphincter, but could be dilated widely without pain. The patient could not relax it voluntarily. The skin of the perineum was normal. In the anterior vaginal wall the fascial sling could be felt, and its position seen, about 1 cm. from the urethral meatus. On flexing and extending the spine, the cord of fascia could be felt to move slightly.

The patient was readmitted for observation on June 4, 1931, five and a half months after her first, and four and a half months after her second, operation. She complained of slight leakage of urine at times, and of pain when the bladder was full, just before, but not during micturation. Her general condition was excellent. The operative scars were healthy; there was slight keloid formation. The fascial transplants felt and acted much the same as on her discharge three months before. The urine was found strongly alkaline with a few pus cells. Cystoscopic examination showed a rather spastic bladder, mildly inflamed at the trigone, but otherwise normal. Renal function was 32 and 18 per cent (total 50 per cent) in two hours. The feces were normal. Leakage of urine was found to be due to carelessness in posture. With some additional coaching, and change of reaction of the urine, there was no further leakage or discomfort, even though the fluid intake was increased to over 3,000 cc. a day. The largest single amount voided was 380 cc. There was no leakage of feces during the six days of observation.

The patient has been followed for over a year, and her control of both urine and feces remains satisfactory.

COMMENT

Congenital absence of the terminal nine or ten segments of the spinal column is an exceedingly rare anomaly. So far we have not been able to find in the literature any reference to a similar condition. Transposition of viscera is doubtless more common among the Chinese than the four reported cases (Fay [4]) would indicate. The case reported here is the fifth.

By definition, a sphincter is a ring of muscle closing an orifice. The term cannot rightly be applied, therefore, either to Wreden's mechanism, or to the one we have described, for they are neither rings nor muscles.

Both in this case and in a subsequent one, we have shared Stone's difficulty in teaching the patient to tighten voluntarily the interlocking loops of fascia about the anus. Moreover, as my colleague Dr. L. M. Ingle has pointed out, contraction of the gluteal muscles brings them nearer the midline and would tend to relax rather than tighten the fascial loops; i. e., the patient might conceivably learn to relax the "sphincter" to some extent, but could not possibly tighten it by any direct muscular action. We believe, therefore, that this ingenious and successful mechanism is not, strictly speaking, voluntary, but that it forms a permanently elastic constriction about the anus, which is sufficient under ordinary conditions to prevent leakage of feces, but which yields to the expulsive force of peristalsis during defecation, without any voluntary relaxation on the part of the patient.

On the other hand, the mechanism which we devised for incontinence of urine in the case just described is truly voluntary.

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4. Fay, T. D.: Report of a Case of Complete Transposition of Thoracic and Abdominal Viscera, Chinese M. J. **46**:318, 1932.

SUMMARY

A Chinese girl, with transposition of thoracic and abdominal viscera and congenital absence of sacrum and coccyx, the latter an exceedingly rare condition, presented problems of complete incontinence of feces and urine, due probably to defective innervation.

Wreden's operation was successfully used to relieve the anal incontinence.

For relief of the urinary incontinence, a method was devised which utilizes bodily posture to tighten or relax a strip of fascia slung about the urethra. In this case it has resulted in voluntary control of the bladder. Details of the operation are given.

The results of the two operations after a period of over a year have been satisfactory.

GYNECOMASTIA

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Gynecomastia, derived from the Greek words: gyne, woman, and mastos, breast, has been a recognized entity since the days of Aristotle,[1] who reported that he had examined several such anomalies. The surgical removal for tumefaction of the breast was first advised by Paulus Aegineta[2] in 1556. He was followed by such men as Haly Abbas[3] and Abul-Casimir el Zahrawi.[4] In 1880, Olphan[5] wrote an interesting article on gynecomastia. Schuchardt,[6] in 1884-1885, reviewed the literature for new tissue formation in the male breast and from his findings published three papers. Two of these dealt with pathologic conditions, while the third described 40 cases as examples of true gynecomastia. Gruber,[7] in 1886, concluded that true gynecomastia was a physiologic phenomenon with all the characteristics of the female organ. Schuchardt,[6] however, included mastitis in his classification.

Gruber,[7] Langer,[8] Luschka[9] and Momberger[10] were some of the first to study the gross and microscopic anatomy of the breast. They

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From the Surgical Pathological Laboratory, Department of Surgery, Johns Hopkins Hospital and University.

1. Aristotle: Hist. Animal (Parisiis), 1542, Calvarini, lib. III, chap. XX.

2. Paulus Aegineta: Medicinae totius enchiridion septem libris universam recte medendi rationem complectus, Opera Basileae, 1556, lib. VI, chap. XLVI, p. 225.

3. Haly Abbas: Liber totius medicine necessaria continens, Lugduni, J. Myteructe, 1523.

4. Abul-Casimir el Zahrawi: Tractatus de operantine manus arabice et latine cura, Oxford, J. Channing, 1778.

5. Olphan, H.: Un mot sur la gynécomastie, ou hypertrophie mammaire chez l'homme, Thèse de Paris, 1880, no. 159.

6. Schuchardt, B.: Zur Casuistik und Statistik der Neubildungen in der männlichen Brust, Arch. f. klin. Chir. **31**:1, 1844; Weitere Mittheilungen zur Casuistik und Statistik der Neubildungen in der männlichen Brust, ibid. **32**:227, 1885; Ueber die Vergrösserung der männlichen Brust, ibid. **31**:59, 1885.

7. Gruber, W.: Ueber die männliche Brustdrüse und über die Gynäcomastie, Mem. Acad. imp. d. sc. de St. Petersburg **10**:1, 1886.

8. Langer, C.: Ueber den Bau und die Entwicklung der Milchdrüse bei den Geschlechtern, Deukschriften d. Kais. akad. d. Wissenchaften math. Naturw. Klasse (Wien) **1**:402, 1852.

9. Luschka, H.: Die Anatomie der männlichen Brustdrüsen, Arch. f. anat. u. Physiol. u. Wissensch. **1**: 402, 1852.

10. Momberger, H.: Untersuchungen über Sitz, Gestalt und Färbung der Brustwarze nebst einigen Bemerkungen über die Contrationsfähigkeit des Warzenhofes und über die in demselben eingelagerten Talgdrüsen, Inaug. Dissert., Giessen, W. Keller, 1860.

maintained that although the mode of development, up to puberty, was essentially the same, the degree of enlargement was less in the male. Kölliker,[11] from his studies, stated that the formation of glandular tissue in the male is more extensive than in the nullipara of the same age. Stieda [12] considered gynecomastia as a hyperplasia of the normal tissues of the male, more marked in the connective tissue element.

Among the modern authors, Deaver and McFarland,[13] Hammett,[14] Kriss,[15] von Gusnar,[16] Steinach [17] and others have made valuable contributions to the literature on this subject.

The two conflicting theories of the causation of gynecomastia are the theory of Halban [18] and that of Herbst [19] and Steinach.[17] Halban [18] maintained that sex characteristics are unisexually determined at fertilization and that the gonads exercise only a protective influence over such characteristics. Herbst [19] and Steinach [17] believe that the anlage of the sex characteristics is bisexual and that the sex hormone has a specific as well as an antagonistic effect.

However, the nature of gynecomastia and the factors entering into the production of this abnormality in the male breast are not clearly understood. One of the reasons for this obscurity is undoubtedly the variety of forms which this disease may take and the multiple conditions which may be associated with it. Much of the confusion has resulted from a failure to define clearly the limits of gynecomastia. The assumption of many authors that mere enlargement in the region of the male breast is synonymous with abnormal physiologic hypertrophy

11. Kölliker, cited by Schaumann, H.: Beitrag zur Kenntnis der Gynäcomastie, Verhandl. der phys.-med. Gesellsch. zu Würzburg **28**:1, 1894.

12. Stieda, H.: Beitrag zur histologischen Kenntnis der sogenannten Gynäkomastie, Beitr. z. klin. Chir. **14**:179, 1895.

13. Deaver, J. B., and McFarland, J.: The Breast; Its Anomalies, Its Diseases and Their Treatment, Philadelphia, P. Blakiston's Son & Company, 1917.

14. Hammett, F. S.: Gynecomastia, Endocrinology **4**:205, 1920.

15. Kriss, B.: Negation of Theory that Gynecomastia is Caused by Cessation of Inhibitory Action of Testicles on Mammary Gland, Arch. f. Gynäk. **141**:503, 1930.

16. von Gusnar, K.: Histologic Study of Male Mammary Glands with Explanation of Certain Pathologic Changes, Arch. f. klin. Chir. **153**:253, 1928.

17. Steinach, E.: Willkürliche Umwandlung von Saügetiermannchen in Tiere mit angepräght weiblichen Geschlechtscharakteren und weiblichen Psyche, Arch. f. d. ges. Physiol. **144**:71, 1912; Geschlechlstrieb und echt sekundäre Geschlechlsmerkmale als Folge der innersekretorischen Funktion der Keimdrüsen, Zentralbl. f. Physiol. **24**:551, 1910.

18. Halban, J.: Die innere Secretion von Ovarium und Placenta und ihre Bedeutung für die Function der Milchdrüse, Arch. f. Gynäk. **75**:353, 1905; Arch. f. Gynäk. **70**:205, 1902; Monatschr. f. Geburtsh. **53**:190, 1904; Arch. f. Gynäk. **114**:289, 1920; ibid. **130**:415, 1927.

19. Herbst, C.: Formative Reize in der tierischen Ontogenese, Leipzig, A. Georgi, 1901.

has also added to the problem. It is therefore necessary to point out that various neoplasms producing enlargement in the tissue surrounding the male nipple have been needlessly included in the discussion.

In general, gynecomastia is the result of (1) general and of (2) local factors. Among the general factors are: (*a*) A lack of sex differentiation in the sex organs (bisexuality). Here the female factor predominates. (*b*) A lack of the male sexual influence where the male factor is lessened and other endocrine disturbances influencing by stimulation or inhibition the male or female hormone. Among the local factors are all local causes producing tumefaction of the male breast (trauma, mastitis, etc.).

GENERAL FACTORS

In support of the first classification, or the lack of sex differentiation in the sex organs, there is the work of Brambell,[20] who discovered ovarian tissue on the surface of the testes of pigs. There is also evidence that gynecomastia has been noted in hermaphrodism and pseudohermaphrodism. These facts are well illustrated by the case of von Humboldt, quoted by Haeckel,[21] of a solitary settler in a South American forest whose wife died at childbirth. In despair the man laid the child over his own breast, and the continued stimulus of suckling movements revived the activity of the mammary glands. Cases of castrated male sheep and goats suckling their offspring have been reported. Humboldt and Franklin [22] Schmetzer,[23] and others have cited examples of gynecomasts who have nursed infants. It may be interesting to note that infants have thrived on "witch's milk," although Schmetzer [23] emphasized that its composition is not that of pure milk. Chinagawayo, a Zulu chief, and a gynecomast at 55 years, had forty wives and over a hundred children, some of which he nursed himself.[24]

There are cases, however, which demonstrate that a gynecomast can have a normal libido. Pétrequin [25] reported a case in which the patient

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20. Brambell, F. W. R.: The Histology of an Hermaphrodite Pig, and Its Developmental Significance, J. Anat. **63**:397, 1929.

21. Haeckel, Ernst: The Evolution of Man, London, K. Paul, Trench & Co., 1883, p. 113.

22. Humboldt and Franklin, cited in Gould, G. M., and Pyle, W. L.: Anomalies and Curiosities of Medicine, Philadelphia, W. B. Saunders Company, 1897, p. 397.

23. Schmetzer: Milchabsonderung in männliche Brüsten, Schmidt's Jahrb. **15**:204, 1837.

24. Shufeldt, R. W.: Gynecomasty, with the Description of a Remarkable Case, Md. Council, Philadelphia **15**:244, 1910.

25. Pétrequin, H.: Existence de trois mamelles chez un homme qui devient père de cinq enfants trimammés, Gaz. méd. de Paris **5**:197, 1837.

(a man) married and proved his potency. Coe,[26] Paulicky [27] and others [28] reported cases of gynecomastia in which there was no evident abnormality of the genitalia and apparently no disturbance or defect in the sex organs. Pseudohermaphrodism, when the sex organs were imperfectly formed and the individual developed secondary female characteristics, was reported by Polaillon,[29] Dennis [30] and others.[31] Beau [32] and Luke [33] likewise cited cases in which gynecomastia was associated with imperfect development of the sex organs.

From the foregoing data the evidence indicates that the ovary and testicle coexist in the embryo, and that in certain cases, because of some developmental injury or anomaly, the testicle is not able to exert its normal inhibitory influence. When this action is withdrawn and the female hormone is inserted, as Steinach [17] demonstrated when he transplanted ovaries in castrated males, there is a tendency toward the formation of female characteristics. Likewise, Halban,[18] Kehrer,[34] Hegar,[35] and others have shown that in females, if the ovaries are removed, the mammae fail to develop, but if again implanted the mammae will resume their normal function. Foges [36] showed that the removal of ovaries, after puberty, leads to a termination of the menstrual cycle and an atrophy of the breasts.

In the second group of gynecomasts, the male sexual influences are lacking. These are the cases of gynecomastia associated with removal of the prostate or testicle or associated with testicular injury. Gynecomastia

26. Coe.: Ann. de gynec. **31**:456, 1889.

27. Paulicky: Deutsche mil.-ärztl. Ztschr., 1882, p. 222.

28. Foot: Remarks on Gynecomazia, Dublin Q. J. M. Sc. **41**:451, 1866; Gynecomastia in a Young Boy, M. Times & Gaz. **1**:11, 1860. Koster: Fall von Gynaekomastia unilateralis, München. med. Wchnschr. **52**:725, 1905. Morgan: Lancet **2**:767, 1875. Scheiber, S. H.: Hypertrophirte Brustdrüsen bei einen 45 jährigen Manne, Med. Jahrb., 1875, p. 261.

29. Polaillon: Sur un cas d'hermaphrodisme, Bull. Acad. de méd., Paris **25**: 557, 1891.

30. Dennis, F. S.: System of Surgery, Philadelphia, Lea Brothers & Co., 1896, vol. 4, p. 942.

31. Chambers: Tr. Obst. Soc., London **21**:256, 1879. Kochenburger: Die Missbildung der weiblichen Genitalien; Casuistischer Beitrag, Ztschr. f. Geburtsch. u. Gynäk. **26**:73, 1893.

32. Beau: Gaz. d. hôp., 1849, p. 563.

33. Luke: M. Times & Gaz. **1**:11 (Jan. 7) 1860.

34. Kehrer, F. A.: Ueber gewisse synchrone nerven erscheinung und cycklische Vorgänge in den Genitalien und anderen Organen, Beitr. z. Geburtsh. u. Gynäk. **4**:228, 1901.

35. Hegar, A.: Korrelationen der Keimdrüsen und Geschlechtstimmung, Beitr. z. Geburtsh. u. Gynäk. **7**:201, 1905.

36. Foges, A.: Zur physiologischen Beziehung zwischen Mamma und Genitalien, Zentralbl. f. Physiol. **19**:233, 1905.

following prostatectomy was reported by Oppenheimer [37] and others.[38] Zeleneff [39] reported a right-sided orchidectomy with hypertrophy of the left breast in a youth 18 years of age. Tellgmann [40] cited a case in which the loss of the testes preceded the development of gynecomastia. Gynecomastia resulting from the disorders of secretion of the testicles has been reported by Bitny-Schliachto.[41] Bailey [42] called attention to the relationship between tumefaction of the mammary glands and mumps. Hassler [43] and others [44] have also reported such cases. Gynecomastia following trauma to the testicle has been recorded by Monaschkin.[45] He found that sections of the breast showed hypertrophy of the ducts, while those of the tumor showed embryonal cells. Gorham [46] and others [47] also reported cases in which atrophy of the testicle and gynecomastia followed trauma.

Simple atrophy of the testicle with gynecomastia has been pointed out by Gorringe [48] and others.[49]

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Orchitis and varicocele producing testicular atrophy and associated with gynecomastia were reported by Schmit [50] and Martel,[51] respectively. The association of varicocele with gynecomastia was found in one case of the present series (P. N. 38637). Galliet [52] reported a case of gynecomastia following a tumor of the epididymis. In these cases the theory of Moszkowicz,[53] that testicular secretion inhibits the growth of the male breasts, is supported. An interesting case of a 19 year old boy suffering from gynecomastia for four years was reported by Schereschewsky.[54] He tells that a year previous to examination this patient was operated on for varicocele at which time a testicular graft, from a 40 year old man, was tried with no effect. The relationship between gynecomastia and chorio-epithelioma of the testis has been stressed and illustrated by case reports by Kriss [15] and others.[55] Gynecomastia developing in cases of teratoma of the testis was reported by Bailey [42] and Cairns.[56] Gynecomastia following sarcoma of the testicle was noted in one case of the present series (P. N. 37777). Erdheim,[57] after examining microscopically the breasts and testicles at autopsy, concluded that the etiology of gynecomastia is closely connected with the causation of sex characteristics and that it could not be attributed to one single factor.

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mazia and Other Aberrations in the Development of Sex, ibid. **3**:327, 1891-1892. Laurent, E.: Les bisexués: Gynécomasties et hermaphrodites, Paris, G. Carré, 1894; De L'hérédité des gynécomastes, Ann. d'hyg. **24**:43, 1890. Lembert: Thèse de Paris, 1878; Gaz. hebd. méd., Sept. 14, 1877. Paulicky: Deutsche mil.-ärztl. Ztschr., 1882, p. 222. Weber, C.: Normiod ridge, Entwickelung beider Brustdrüsen bei Manne, Ztschr. d. deutsch. Chir. Ver. **5**:336, 1852.

50. Schmit, C.: Cong. franç. de Chir., Paris **6**:284, 1892; Deux cas de gynécomastie développés sans cause appréciable, Rec. de mém. de méd. mil., Paris **38**:690, 1881.

51. Martel: Hypertrophie du sein gauche chez un homme atteint de varicocèle du même côte, Arch. de méd. nav., Paris **60**:152, 1893.

52. Galliet: Sur deux cas de coincidance du développement anormal de la mamelle chez l'homme, avec une tumeur de l'épididyme, Compt. rend. Soc. de biol. **2**:36, 1850.

53. Moszkowicz, L.: Ueber den monatlichen Zyklus der Brustdrüse, Arch. f. klin. Chir. **142**:374, 1926.

54. Schereschewsky, N. A.: Sur la pathogénie et le traitement de la gynécomastie, Rev. franç. d'endocrinol. **6**:57, 1928.

55. Herzenberg, H.: Gynecomasty: Its Special Relation to Chorio-Epithelioma in Man, Virchows Arch. f. path. Anat. **263**:781, 1927. Prym, P.: Chorio-Epithelioma in a Man with Gynecomastia, Beitr. z. path. Anat. u. z. allg. Path. **85**:703 (Nov. 20) 1930.

56. Cairns, H. W. B.: Neoplasms of the Testicle, Lancet **1**:845 (April 24) 1926.

57. Erdheim, S.: Ueber Gynäkomastia, Deutsche Ztschr. f. Chir. **181**:208, 1928.

A thorough study on the relationship of the testicles to gynecomastia was made by Kriss,[15] who castrated twenty male guinea-pigs and afterward examined their breasts microscopically. In all of his cases he found no change in the glandular tissue. Further evidence that the testicles alone have little influence on gynecomastia was obtained by Stein and Herrmann,[58] who injected corpus luteum in castrated and noncastrated rabbits and found that both developed enlargement of the mammary glands. This work, however, only proves that the testicles, in themselves, are not the lone influence that prevents an enlargement of the mammary tissue. The testicles are closely connected to the intricate system of endocrine glands, and although they probably play the greatest part in sexual development, they function as a correlated part of the endocrine system.

The effects of other endocrine disturbances are shown by the association of gynecomastia with pituitary, suprarenal, cortical and thyroid disturbances.

The hypophyseal origin of Fröhlich's syndrome was definitely established by Cushing [59] and Ashner,[60] who demonstrated that obesity and genital hyperplasia developed in hypophysectomized animals. They also stated that the adiposity was caused by a deficiency of the posterior lobe. Cushing [59] explained that hyperplasia of the anterior lobe stimulates tissue growth, especially in the skeletal, cuticular and subcuticular tissues; conversely, when there is an insufficiency of the anterior lobe skeletal growth and sexual development are inhibited. Hyperplasia of the posterior lobe is associated with metabolism, for when this lobe is rendered inactive by disease or compression, metabolism is checked and an acquired high tolerance for carbohydrates permits the storage of fat.

Evidence of hypopituitarism, in a boy of 14 years, associated with mammary enlargement and small genitalia, was recorded by Gibson.[61] Among the evidence of hyperfunction of the anterior lobe, associated with mammary hypertrophy, is the report, by Roth,[62] of acromegalia in a man whose mammae secreted true milk. Hanel [63] described a case of a

58. Herrmann, E., and Stein, M.: Ueber künstliche Entwicklungschemmung männlicher sekundärer Geschlechtsmerkmale, Arch. f. Entwicklungsmechn. **48**:447, 1921.

59. Cushing, H.: Surgical Experiences with Pituitary Disorders, Weir Mitchell Lecture, J. A. M. A. **63**:1515, 1914.

60. Ashner, B.: Demonstrierte hypophysektomierte Hunde, Wien. klin. Wchnschr. **22**:1730, 1909.

61. Gibson, H. J. C.: Case of Exaggerated Gynecomastia, Edinburgh M. J. **30**:668, 1923.

62. Roth, O.: Auftreten von Milchsekretion bei einem an Akromegalie leidenden Patienten, Berl. klin. Wchnschr. **55**:305, 1918.

63. Hanel, H.: Mama lactans persistens masculina, Klin. Wchnschr. **5**:386, 1918.

43 year old man having possessed lactating breasts at 21 years, who later became the father of two normal children. Subsequent to his death, autopsy revealed a malignant disease of his hypophysis. Moehling [64] performed an operation on a 52 year old man who had developed gynecomastia in association with a pituitary adenoma. Mammary hypertrophy associated with attacks of chorea minor was noted by Weber.[65] Siegmund and Mahnert [66] demonstrated the effect of the anterior lobe hormone on the development of the sex glands in infants and fetuses. Intraperitoneal injections of an extract from the anterior lobe of the hypophysis, which were given by Putnam, Teel and Benedict,[67] produced an increase in the size of the nipples of a bulldog. This was also demonstrated by Frank and Unger (Scherschewsky [54]). Davidoff [68] called attention to a case of acromegalia in which lactation and menorrhagia had persisted for five years. A report from the woman's surgeon, who had performed a laparotomy two years previously, said that both ovaries were in a state of semi-atrophy with no signs of ovulation.

The concurrence of cirrhosis of the liver and gynecomastia, or tumors of the male breast, has been reported by d'Antona [69] and others.[70] This may be explained on the basis of a mild form of Wilson's disease (tetanoid chorea) in which there is a degeneration of the lenticular nucleus associated with hepatic cirrhosis. It is interesting to note that in d'Antona's case there was testicular atrophy.

There are cases in which the disturbance of the suprarenal cortex are associated with gynecomastia. Mathias,[71] at postmortem examination, described a carcinoma of the suprarenal cortex associated with

64. Moehling, R. C.: Pituitary Tumor Associated with Gynecomastia, Endocrinology **13**:529 (Nov.-Dec.) 1929.

65. Weber, F. P.: Notes on Mammary Enlargement, from Endocrine Disturbances in Males, M. Press **126**:425 (Nov. 21) 1928.

66. Siegmund, H., and Mahnert, A.: Effect of Hormone of Anterior Lobe of Infants and Fetuses on Development of Infantile Sex Glands, München. med. Wchnschr. **75**:1835 (Oct. 26) 1928.

67. Putnam, T. J.; Teel, H., and Benedict, E. B.: The Preparation of a Sterile Active Extract from the Anterior Lobe of the Hypophysis, Am. J. Physiol. **84**:157, 1928.

68. Davidoff, L. M.: Studies in Acromegaly, Endocrinology **10**:477, 1926.

69. d'Antona, L.: Hepatic Cirrhosis, Tumefaction of Male Mammary Glands and Testicular Atrophy, Policlinico (sez. prat.) **38**:261 (Feb. 23) 1931.

70. Paula, F.: Concurrence of Cirrhosis of the Liver and Gynecomastia; Four Cases, Deutsches Arch. f. klin. Med. **169**:83, 1930. Pellegrini, A.: Male Breast: Hypertrophy in Hepatic Cirrhosis, Semana méd. **1**:1108 (May 5) 1927. Tattoni, A.: Male Breast: Hypertrophy in Hepatic Cirrhosis, Morgagni **69**:1081 (July 10) 1927.

71. Mathias, E.: Ueber Geschwülste der Nebennierenrinde mit morphogenetischen Wirkungen, Virchows Arch. f. path. Anat. **236**:446, 1922.

mammary enlargement. Weber [72] recorded a similar case at a later date. A case of pseudohermaphrodism with suprarenal hyperplasia has been described by Brutschy.[73] Evidence that the injection of mammary extract producing a hypertrophy of the suprarenal gland and arrest in development of the testicle has been established by Spirito,[74] who, in addition, sanctions this extract in the treatment of Addison's disease.

A hyperthyroidosis accompanied by enlargement of the mamma was noted by Freeman.[75] At a later date, Alexander [76] called attention to a case of myxedema following mammary hypertrophy in childhood. In the present series, case P. N. 37676 was associated with hyperthyroidism, and P. N. 39356, with myxedema.

These cases clearly indicate the close relationship of the endocrine system to gynecomastia. A logical deduction indicates that the endocrine glands influence the male and female hormone activity by stimulation or retardation.

LOCAL FACTORS

Gynecomastia may be unilateral or bilateral, 87.2 per cent being unilateral and 12.8 per cent being bilateral. The hypertrophy which occurs may be caused by general or local factors or both. The local factors include some forms of chronic irritation which seem to create ideal conditions for the activation of cell growth. Although trauma has been associated with gynecomastia by Deaver and McFarland,[13] Léon [77] and others,[78] it occurs in only 14.5 per cent of the cases. It is questionable whether trauma plays any particular rôle in this phase of activity, although it is probably more closely associated with unilateral growth. The presence of some form of chronic irritation is suggested by the eosinophils and plasma cells in the peritubular, round cell infiltra-

72. Weber, F. P.: Cutaneous Striae, Purpura, High Blood Pressure, Amenorrhoea and Obesity of the Type Sometimes Connected with Cortical Tumors of the Adrenal Glands, Occurring in the Absence of Any Such Tumour, Brit. J. Dermat. **38**:1, 1926.

73. Brutschy, P.: Hochgradige Lipoidhyperplasie beider Nebennieren mit herdförmiger Kalkablagerung bei einen Fall von Hypospadiasis penis-scrotalis und doppelseitigem Kryptorchismus mit unechter akzessorischer Nebenniere am rechten Hoden, Frankfurt. Ztschr. f. Path. **24**:203, 1920.

74. Spirito, F.: Correlazioni fuzionali surrenomammare. Proposta di un nuovo metodo di cura del morbo di addison, Rassegna di clin., terap. e sc. aff. **22**:265, 1923.

75. Freeman, J. K.: Hyperthyroidosis Associated with Gynecomastia, Therap. Gaz. **40**:9, 1916.

76. Alexander, W.: Myxedema Following Mammary Hypertrophy in Childhood, Brit. M. J. **1**:349 (Feb. 23) 1929.

77. Léon: Arch. de méd. nav. Paris **31**:213, 1879.

78. Nélaton, A.: Gaz. d. hôp. **29**:126, 1856. Peters, D. C.: Hypertrophy of the Mammary Gland in a Soldier, Am. M. Times, N. Y. **6**:196, 1863.

tion, which is seen in practically every form of gynecomastia. Tenderness in 50 per cent of the cases also suggests a source of irritation. The round cell infiltration seen in gynecomastia has been explained by Dietrich [79] and Consten [80] as a resorptive process which may be similarly seen after physiologic hypertrophies and in the regressive phases of the female breast. However, the infiltration is often seen in hypertrophies of short duration before any evidence of resorption is noted. The presence of eosinophils and plasma cells is sometimes so marked that microscopically the hyperplasia simulates mastitis. In some cases

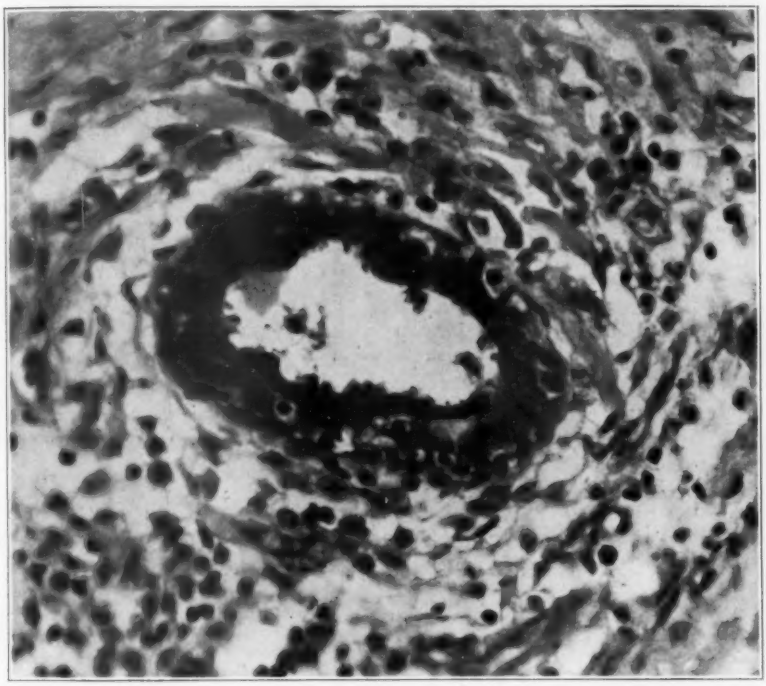

Fig. 1 (path. no. 41316).—Microscopic structure of tissues from a white youth, 17 years of age, who, for ten days, complained of a slightly painful tumor of his breast. The mass was excised and the patient is well two years later. The section shows epithelial hyperplasia surrounded by young fibroblasts interspersed with polymorphonuclear leukocytes, lymphoid and plasma cells. The section illustrates the association of hypertrophy with mastitis.

there is definitely an association with mastitis (fig. 1). Ciöffi [81] regarded tumefaction at the age of puberty in males as a temporary mastitis

79. Dietrich and Frangenheim: Neue Deutsche Chir. Stuttgart, Ferdinand Enke, 1926, vol. 35.

80. Consten, A.: Diffuse Fibromatosis of Mamma in Man, Deutsche Ztschr. f. Chir. **167**:264, 1921.

81. Ciöffi, A.: Tumori mammari maschile, Ann. ital. di chir. **9**:1147, 1930.

Path. No.	Age	Color	Symptoms	Duration, Months	Tumor	Location	Treatment	Microscopic Examination	Results
45537	42	C	.....	..	Unilateral; discrete	.....	Excision of breast	Fibrous hyperplasia	
45411	43	C	Disappearing, painless lump	24	Unilateral; discrete	Right	Excision of tumor	Fibrous hyperplasia; atrophy of duct cell	
44224	23	W	Trauma; mass, tender; gradual growth	36	Unilateral; discrete	Left	X-rays, nine exposures; excision of tumor one year later	Duct and marked fibrous hyperplasia (stroma)	Well 1 yr. later
43911	..	..	.....	..	.....	.....	Amputation of breast	Duct and fibrous hyperplasia (stroma)	Lost 3 yrs. later
42730	9	..	Gradual enlargement, tender	1½	Unilateral; discrete	.....	Excision of tumor	Fibrous and marked duct hyperplasia (periductal)	Well 1 yr. later
42294	..	W	Trauma; slow growing mass	1½	Bilateral; discrete	Right and left	Complete excision of both breasts	Duct and fibrous hyperplasia	Well 2 yrs. later
42222	55	W	Tumor; rapid growth, tender	2	Unilateral; discrete	Left	Excision of tumor	Marked duct and fibrous hyperplasia (periductal)	Well 2 yrs. later
41925	12	W	Tumor; slow growth, painful	¾	Unilateral; discrete	Left	Excision of tumor	Duct and fibrous hyperplasia (periductal)	Well 4 yrs. later
41894	63	W	Trauma; tumor, slow growth; pain	1½	Unilateral; discrete	Left	Excision of left breast	Duct and fibrous hyperplasia (stroma)	Well 2 yrs. later
41657	34	C	.....	..	.....	.....	Excision of breast	Duct and fibrous hyperplasia (stroma)	Lost 4 yrs. later
41316	17	W	Tumor; pain	⅓	Unilateral; discrete	.....	Partial excision of tumor	Marked duct and fibrous hyperplasia (periductal)	Lost 2 yrs. later
41077	14	W	Trauma; tumor; pain	¾	Unilateral; discrete	Left	Excision of tumor	Marked duct and fibrous hyperplasia (stroma)	Lost 4 yrs. later
40668	67	W	Tumor; tender	1½	Unilateral; discrete	Right	Complete excision of breast; one roentgen treatment	Duct and marked fibrous hyperplasia (periductal)	Dead 1 yr. later; bladder inflammation
40630	62	W	Tumor; pain	2½	Unilateral; discrete	Right	Complete excision of breast; cautery	Duct and fibrous hyperplasia (periductal)	Well 3 yrs. later
39909	45	W	.....	..	.....	Left	Excision of breast	Duct and marked fibrous hyperplasia (stroma)	
39658	34	W	Tumor	..	Unilateral; diffuse	.....	Excision of breast	Fibrous hyperplasia; duct dilatation	Well 4 yrs. later
39619	44	W	Tenderness; tumor	4	Unilateral; discrete	Left	Roentgen treatment; excision of breast 1 month later	Fibrous hyperplasia (stroma); duct cell atrophy	Lost 5 yrs. later
39394	55	W	Tenderness; tumor	3	Unilateral; discrete	Left	Amputation of breast	Duct and fibrous hyperplasia (stroma)	Well 1¼ yrs. later
39356*	43	W	Tumor; pain	¾	Unilateral; discrete	Left	Excision of breast	Duct and fibrous hyperplasia; duct dilatation	Well 4 yrs. later (hypothyroidism)
38637*	20	C	Trauma; diffuse enlargement	96	Unilateral; diffuse; (one discrete nodule)	Left	Excision of tumor	Duct and fibrous hyperplasia	Lost 5 yrs. later

38487	55	W	Tumor; slow growth; painful	..	Unilateral; discrete	.....	Excision of tumor	Duct and fibrous hyperplasia	Lost 5 yrs. later
38464	33	W	Pain; gradual swelling	2½	Unilateral; discrete	Right	Excision of tumor	Marked duct and fibrous hyperplasia (periductal)	Living 9 yrs. later (tuberculosis ?)
37777*	32	W	Tumor	1	Unilateral; discrete	Right	Excision of tumor	Marked duct and fibrous hyperplasia (periductal)	Well 6 yrs. later
37676*	51	W	Tumor; tender	2	Bilateral; discrete	Right and left	Excision of tumor masses	Duct and fibrous hyperplasia	Well 5 yrs. later
37493	70	W	Tumor; tender; slow growth	1½	Unilateral; discrete	Left	Excision of tumor	Duct and marked fibrous hyperplasia (periductal)	Lost 6 yrs. later
37379	14	W	Tumor; slow growth	5	Unilateral; discrete	Left	Excision of tumor	Marked duct and fibrous hyperplasia (stroma)	Lost 6 yrs. later
37203	19	W	Trauma; tumor; pain	4	Unilateral; discrete	Left	Excision of tumor	Duct and fibrous hyperplasia (periductal)	Well 6 yrs. later
37046	45	W	Tenderness; tumor slow growth	2½	Unilateral; (discrete); diffuse	Right	Complete excision of breast	Duct and marked fibrous hyperplasia (stroma); cystic dilatation	Well 6 yrs. later
37042	17	W	Bilateral enlargement	2	Bilateral; diffuse	Right and left	Bilateral, complete amputation	Duct and marked fibrous hyperplasia; cystic dilatation	Lost 6 yrs. later
36879	36	C	Tumor; slow growth; pain	12	Unilateral; multiple; discrete	Left	Amputation	Dense fibrous stroma	Lost 6 yrs. later
36606	..	..	Trauma; tumor; tender	1	Unilateral; discrete	Right	Complete excision of breast	Duct and fibrous hyperplasia; duct dilatation	Well 4 yrs. later
36144	27	C	Tumor; slow growth; pain	8	Unilateral; discrete	.....	Excision of tumor	Duct and fibrous hyperplasia (stroma)	Well 7 yrs. later
36022	43	W	Tumor; gradual growth; pain	2	Unilateral; discrete	.....	Complete amputation of breast	Duct and fibrous hyperplasia (stroma)	Well (lump in other breast) 1 yr. later
35916	39	..	.....	..	Unilateral; multiple	.....	Excision of tumor masses	Duct and fibrous hyperplasia (periductal)	Dead 1 yr. later
36768	48	W	Tumor; tender	1	Unilateral; diffuse; discrete nodules	Right	Complete excision of breast	Duct and marked fibrous hyperplasia (periductal)	Dead 5 yrs. later; generalized arthritis; no recurrence
35524	28	W	Tumor	3	Unilateral; discrete	Left	Complete excision of breast	Marked duct and fibrous hyperplasia	Well 7 yrs. later
35350	73	W	Tumor	3	Unilateral; discrete	.....	Amputation of breast	Duct and fibrous hyperplasia (stroma)	Lost 7 yrs. later
33816	61	W	Tumor; short duration	..	Unilateral	Right	Excision of breast	Marked duct and fibrous hyperplasia (periductal)	Lost 7 yrs. later
32768	12	W	Tumor; slow growth; pain	7	Unilateral; diffuse	Right	X-ray, three series	(No operation)	Well 8 yrs. later; tumor disappeared
32274	42	W	Tumor	2	Unilateral	.....	Excision of tumor	Duct and fibrous hyperplasia	Well 8 yrs. later; no recurrence
31965	29	C	Tumors; pain	..	Unilateral; multiple; discrete	Left	Excision of breast	Duct and marked fibrous hyperplasia (stroma)	Lost 9 yrs. later
31661	33	W	Tumor	3	Unilateral; discrete	Left	Excision of tumor	Fibrous hyperplasia; duct dilatation	Lost 8 yrs. later
31660	63	W	Pain; tumor	3	Unilateral; discrete	Left	Excision of tumor	Fibrous hyperplasia; duct dilatation	Lost 8 yrs. later

* The case was complicated by one of the following conditions: hyperthyroidism, hypothyroidism, varicocele and carcinoma of the testicle.

Gynecomastia—Continued

Path. No.	Age	Color	Symptoms	Duration, Months	Tumor	Location	Treatment	Microscopic Examination	Results
30834	65	W	Pain; tumor	2	Bilateral; diffuse; (one discrete nodule)	Right and left	Bilateral amputation of breast	Duct and fibrous hyperplasia	Died 5 yrs. later
30542	68	W	Tumor	3	Unilateral; diffuse	Left	Complete excision of breast	Duct and fibrous hyperplasia	Well 8 yrs. later
30510	15	W	Tumor	1	Unilateral; discrete	Left	Excision of breast	Duct and fibrous hyperplasia	Well 9 yrs. later; no recurrence
30218	..	W	Nipple irritation	1¼	Unilateral	.....	Excision of nipple and areola	Duct and fibrous hyperplasia (stroma)	Dead 5 yrs. later
29414	20	W	Tenderness; tumor	24	Unilateral; multiple and discrete	Right	Observed	.....	Well 10½ yrs. later; no change in tumor mass
29025	74	W	Burning; tumor	3	Unilateral; discrete (similar mass in opposite breast 5 yrs. later	Right	Excision of tumor; excision of second tumor 5 yrs. later	Fibrous hyperplasia; duct hyperplasia with dilatation	Dead 3 yrs. later; heart trouble
28810	..	W	Tumor; tender	..	Unilateral; discrete	.....	Excision of tumor	Duct and marked fibrous hyperplasia (periductal)	Lost 9 yrs. later
25908	26	W	Tumor; both breasts; tenderness	36	Bilateral; discrete	Right and left	Observed; later, amputation of right breast	Dense fibrous stroma	Lost 10 yrs. later
24824	22	W	Trauma; tumor	72	Unilateral; diffuse; discrete nodule	Right	Excision of breast	Duct and fibrous hyperplasia (periductal)	Well 12 yrs. later; no recurrences
24617	48	W	Yellow discharge; tumor; slow growth	36	Unilateral; discrete	Right	Excision of breast; cautery	Papillomatous duct hyperplasia	Well 8 yrs. later
24430	16	W	Tumor; pain	2	Unilateral; discrete	Right	Excision of breast	Marked duct and fibrous hyperplasia	Well 11 yrs. later
24174	36	W	Trauma; tumor	12 yrs.	Unilateral; discrete	Right	Excision of breast	Dense fibrous stroma	Well 12 yrs. later
23501	68	W	Tumor; slow growth	7	Unilateral; discrete	Right	Excision of tumor	Duct and fibrous hyperplasia (stroma)	Well 13 yrs. later
23500	33	C	Tumor; pain	36	Unilateral; discrete	Left	Excision of breast	Duct hyperplasia (papilloma)	Lost 13 yrs. later
22192	39	W	Tumor; pain	..	Unilateral; diffuse	.....	Amputation of breast	Duct and marked fibrous hyperplasia (periductal)	Lost 14 yrs. later
20499	72	W	Tumor	..	Unilateral; discrete	Left	Excision of tumor	Duct and fibrous hyperplasia	Dead 3 yrs. later; heart failure
14074	33	W	Tumor; hemorrhagic discharge	46	Unilateral; discrete	Right	Excision of tumor	Duct hyperplasia (papillomatous)	Lost
13623	54	W	Tumor	2	Unilateral; discrete	Left	Excision of breast	Duct and fibrous hyperplasia (stroma)	Dead 10 yrs. later; pneumonia
12380	40	W	Itching; tumor	3½	Unilateral; discrete	Right	Excision of breast	Duct and fibrous hyperplasia	Lost 18 yrs. later

12302	22	W	Tumor	3	Unilateral; diffuse	Left	Excision of breast	Duct and marked fibrous hyperplasia (periductal); duct dilatation	Lost 19 yrs. later
12007	45	W	Tumor	6	Unilateral; diffuse	Right	Excision of breast	Marked duct and fibrous hyperplasia (periductal)	Well 11 yrs. later
11763	33	W	Pain; tumor; gradual growth; (excision of tumor 6 mos. previously)	12	Unilateral; discrete	Right	Excision of breast	Duct and fibrous hyperplasia (stroma)	Lost 19 yrs. later
10323	64	C	Trauma ?; tumor; pain	4½	Unilateral; discrete	Left	Radical amputation of breast; axilla	Duct and fibrous hyperplasia	Lost
9669	63	W	Tumor	12	Unilateral; discrete	Left	Excision of breast	Duct and fibrous hyperplasia	Lost 22 yrs. later
9516	62	W	Trauma; tumor; pain	3	Unilateral; discrete	Left	Excision of breast	Duct hyperplasia with papilloma formation	Lost 22 yrs. later
7982	19	W	Tumor	70	Unilateral; discrete	Right	Excision of breast	Marked fibrous hyperplasia (stroma)	Lost 25 yrs. later
7621, 7526	40	W	Tumor	1	Unilateral; diffuse	Right	Complete excision of breast	Marked duct and fibrous hyperplasia (periductal)	Well 24 yrs. later
7448	..	..	Tumor	..	Unilateral; localized	.....	Amputation of breast	Duct and fibrous hyperplasia (stroma)	Dead 22 yrs. later; paralysis
7418	45	W	Burning sensation; tumor	½	Unilateral; diffuse	Left	Amputation of breast	Marked duct and fibrous hyperplasia (periductal)	Well 1½ yrs. later
7402	62	W	Tumor; pain	12	Unilateral; multiple; discrete	Left	Excision of breast	Duct and fibrous hyperplasia (stroma)	Lost 26 yrs. later
6317	19	W	Tumor; slow growth	48	Unilateral; discrete	.....	Excision of tumor	Marked fibrous hyperplasia (stroma) duct dilatation	Lost 26 yrs. later
5169	36	W	Tumor	10 yrs.	Unilateral; discrete	Right	Excision of tumor	Fibrous hyperplasia; papilloma	Lost 12 yrs. later
4767	44	W	Amputation right breast (painful hypertrophy); 2½ yrs. later, pain; tumors	1¾	Unilateral; discrete (multiple)	Left	Amputation of breast (left)	Duct and marked fibrous hyperplasia	Lost 27 yrs. later
3869	42	W	Tumors; pain	3	Unilateral; diffuse	Right	Complete amputation of breast	Duct and fibrous hyperplasia	Well 1 yr. later
3329	..	..	Tumor	3	Unilateral; diffuse	.....	Excision of breast	Duct and fibrous hyperplasia (stroma)	Lost
3068	25	W	Tumor	..	Unilateral; diffuse	Left	Excision of breast	Duct and fibrous hyperplasia (stroma)	Dead 30 yrs. later
1842	20	W	Tumor; pain	..	Unilateral; diffuse	Right	Excision of breast	Duct and fibrous hyperplasia (stroma)	Lost
630	60	W	Trauma; tumor; pain	1½	Unilateral; multiple; discrete	Right	Radical amputation of breast	Dense fibrous stroma	Lost
571	47	W	Trauma; pain; tumor	¾	Unilateral; diffuse	Left	Excision of tumor	Duct and marked fibrous hyperplasia (periductal)	Well 37 yrs. later
569	36	W	Tumor	10 yrs.	Unilateral; discrete	Right	Excision of tumor	Marked duct dilatation; (papillomatous)	Lost
J.C.B. no. 6670	39	W	Trauma; areolar scar	..	Unilateral; discrete	.....	Operation not advised	.....	Well 16 yrs. later; no recurrence
Surg. no. 1940	43	W	Tumor; slow growth	36	Unilateral; discrete	Right	Excision of tumor	Marked fibrous hyperplasia	Well 1 yr. 8 mos. after operation
376	21	W	Tumor; pain	12	Bilateral; discrete	Right and left	Excision of tumor (right)	Duct and marked fibrous hyperplasia (stroma)	Lost 8 yrs. later
Autopsy 10078	1½ mos.	W	Tumor	11 days	Bilateral; diffuse	Right and left	.....	.....	Death (pyloric stenosis)

and that occurring in middle-aged and elderly persons as a chronic hypertrophy.

Heredity has been named as a factor in gynecomastia, but the occurrence is so infrequent that it may be classed as incidental. Hutchinson [82] and others [83] have reported such instances. Hereditary influences, however, bring to mind the possibility of inherent atavistic reversion which should not be overlooked in the final analysis of gynecomastia.

PERSONAL STUDY

Statistics on tumors of the male and female breast recorded in the Surgical Pathological Laboratory of the Johns Hopkins Hospital and University reveal that tumors of the male breast occur in 3.6 per cent of the total.

The present study was made from 130 cases of tumors of the male breast collected in the laboratory, the first case being recorded in 1890. Of the 130 cases, 99, or 76.1 per cent, were benign and 31, or 23.9 per cent, were malignant. Of the 99 benign cases, 88 were hypertrophies.

The results of further statistical study of the hypertrophies is shown in the following tabulation:

Race (white), 88%	Location (bilateral), 12.8%
Age (average), 31-50 years	Trauma, 14%
Youngest case, 1½ months	Pain, 30%
Oldest case, 74 years	Tenderness, 50%
Duration (average), 14.7 months	Tumor (discrete), 76%
Duration (extremes), 11 days; 12 years	Tumor (diffuse), 31.5%
Location: left, 47.2%; right, 40%	Result (benign), 100%

A study of the microscopic sections and a correlation of them with the histories, operations and postoperative observations, favor the inclusion of the so-called fibro-adenomas in the classification of gynecomastia. The following case explains and prompted this decision:

A healthy, beardless boy 17 years of age, noted a symptomless, bilateral, symmetrical enlargement of his breasts. For two years he was ridiculed for having feminine breasts, and on one occasion this deformity prevented his admission into the marine corps. The remainder of the history and physical examination were essentially unimportant. For cosmetic effect, a bilateral amputation of the breasts was performed. The gross specimens (figs. 2*A* and *B*) showed a diffuse fibrous hypertrophy. Between the white fibrous strands there were pink semitranslucent and yellow areas. The microscopic picture was typical of so-called fibro-adenoma. There were areas of duct dilatation, which in some places showed early papilloma formation (fig. 3). The close relationship of such fibro-adenomas with the various other phases of hyperplasia found in gynecomastia is brought out in the microscopic description of these lesions subsequently presented.

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82. Hutchinson, J.: Gynecomazia, Arch. Surg. **3**:327, 1891.

83. Savitschky: Case of Gynecomastia, St. Louis M. & S. J. **66**:118, 1894. Schaumann, H.: Beitrag zur Kenntnis der Gynäkomastic, Verhandl. d. phys.-med Gesellsch. zu Würsburg **28**:1, 1894-1895.

HISTOLOGY

The hypertrophy characterizing gynecomastia is infinitely connected with the normal growth of the male breast observed in infancy and at puberty.

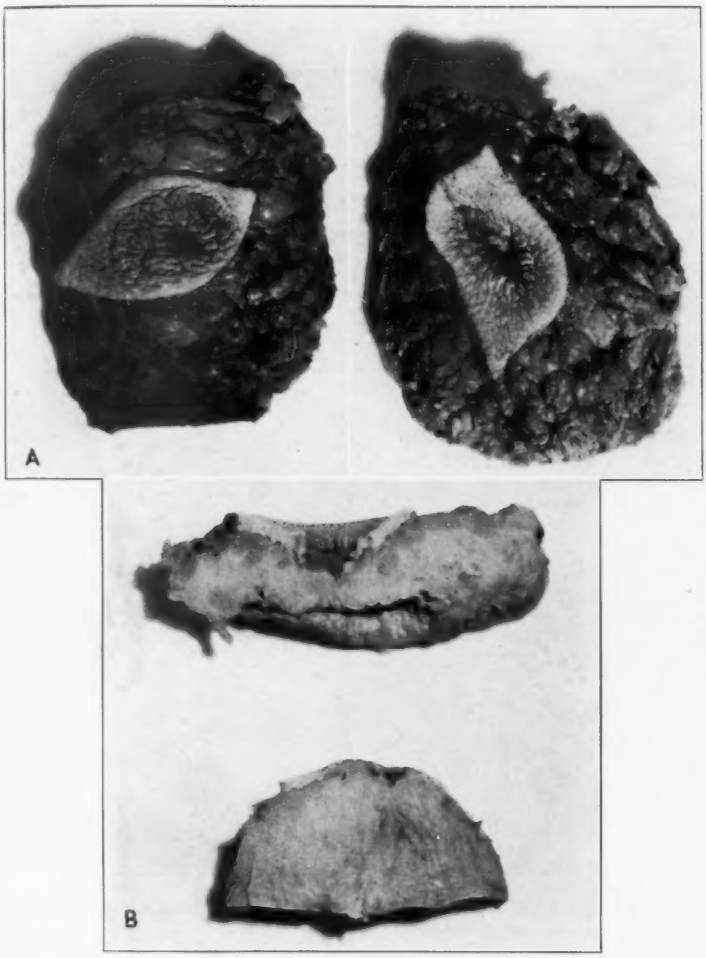

Fig. 2 (path. no. 37042).—*A*, the breasts of a white youth, 17 years of age, which were removed for cosmetic effect because of bilateral symmetrical enlargement. The gross specimens are surrounded by pads of fat with no evidence of glandular tissue (*B*). *B*, cross-section of the breasts showing diffuse, bilateral fibrous hypertrophy which includes pink areas between its strands and a few yellow opacities.

The normal male gland has a tendency to hypertrophy in the first year of life, but after this period development progresses slowly until puberty is reached. At puberty development is at its height. The

parenchyma begins its development as long tubelike processes, descending from the overlying epidermis (fig. 8). The ends of these processes become distended but have no tendency to form lobules. The surrounding stroma is composed of moderately dense fibrous tissue. At the end of the first year of life, and especially at puberty, the parenchyma has a tendency to become hypertrophied and hyperplastic. This hyper-

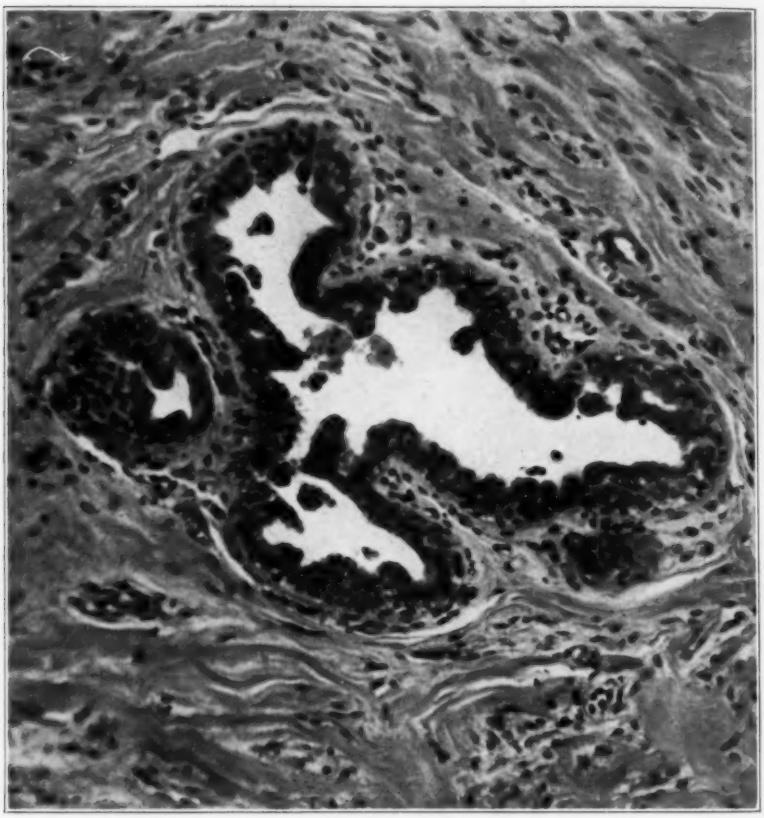

Fig. 3 (path. no. 37042).—Microscopic section of breast tissue in figure 2, demonstrating an epithelial hyperplasia with beginning papillary formation and desquamation into the duct lumen. The periductal fibrous stroma is dense, collagenous and adult in structure.

plasia is accompanied by the development of two or more rows of nuclei which may be either oval or cylindric in shape. The surrounding stroma forms a variable amount of young and loose periductal connective tissue which contains numerous blood vessels. This hypertrophy is not marked in the majority of cases, and as a rule there is no clinical evidence of its presence.

In gynecomastia the extent of the cell proliferation is dependent on the intensity and the duration of the stimulating influence. When gynecomastia is present, the parenchyma and stroma show evidence of hyperplasia which seems to be more pronounced in cases of short duration.

There is an increase in the epithelial lining of the ducts. The ducts are lined with cells containing a central, small, clear nucleus. As the lumen is approached from the periphery, the cells vary from a cuboidal to a cylindric form, the cylindric form predominating when proliferation is most marked. The lumen is often filled with the eosin-staining

Fig. 4 (path. no. 40630).—Microscopic structure of breast tissues from a white man, 62 years of age, who, for two and one half months, complained of stitch-like pains in his breast. This was associated with a small nodule which produced a retraction of his right nipple. The breast was excised, and the patient is living and well three years later. The microscopic section shows epithelial hyperplasia surrounded by the typical, young, loose, periductal, fibrous stroma seen in early cases of gynecomastia.

material of desquamated cells. The periductal stroma is marked by a proliferation of loose connective tissue containing many young fibroblasts and blood vessels (fig. 4). The active growth which is present in this periductal stroma is sometimes indicated by the presence of mitosis (fig. 5). Immediately surrounding the duct there is a lymphocytic infiltration which is accompanied by plasma cells and a few eosinophils.

If the hyperplasia is present for six or more months the proliferation of the parenchyma is not as marked, but the lumen of the duct contains a greater number of desquamated cells. The lining cells vary from an oval to a cuboidal shape (fig. 6). The stroma also changes its appearance, being characterized by a marked condensation of the periductal connective tissue and a decrease in the number of blood vessels.

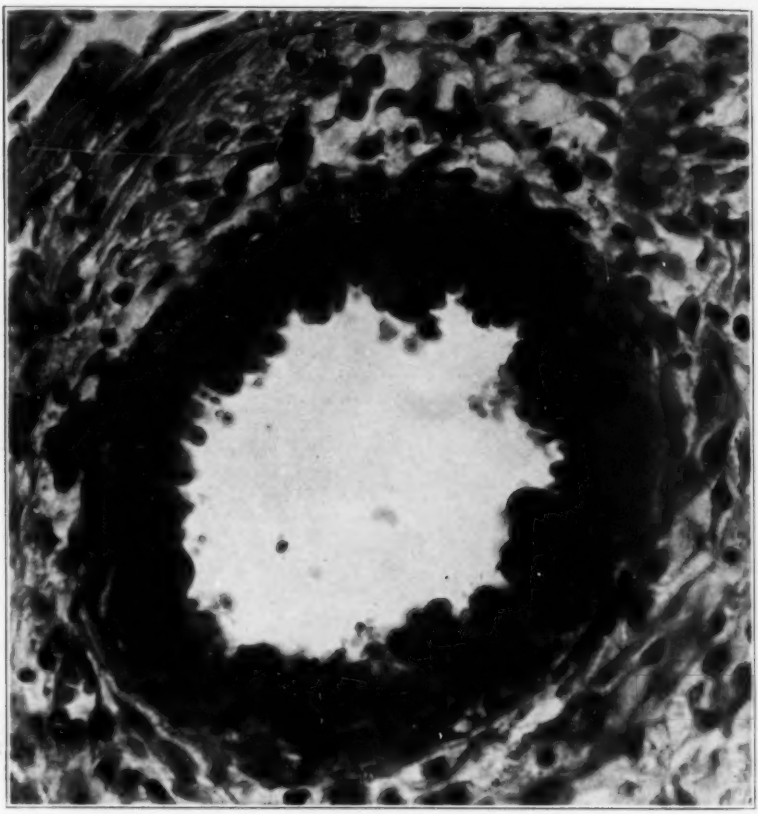

Fig. 5 (path. no. 38464).—Microscopic structure of breast tissue from a white man, 33 years of age, who, for ten weeks, complained of pain and a gradually increasing swelling of his right breast. The tumor was excised, and the patient is living four years later. The microscopic section shows epithelial hyperplasia embedded in a young, fibrous periductal stroma whose rapid growth is indicated by a mitotic figure located immediately above the epithelial lining of the duct.

The hyperplasia of the duct in some cases may take the form of papillomatous proliferation and in later stages may develop into intracystic papillomas (fig. 7). A dilatation of the ducts is frequently seen, but since such dilatations occur in the normal male breast (fig. 8) it is of no great significance.

If a hyperplasia has been present for more than a year, the young periductal connective tissue becomes adult in structure and acquires varying amounts of collagen. A case illustrating such a change is given:

A colored man, 33 years of age, gave a history of having noted a small, tender nodule in his left breast three years previously. After being quiescent for approximately two and a half years, the nodule suddenly became painful and increased in size, two weeks previous to examination.

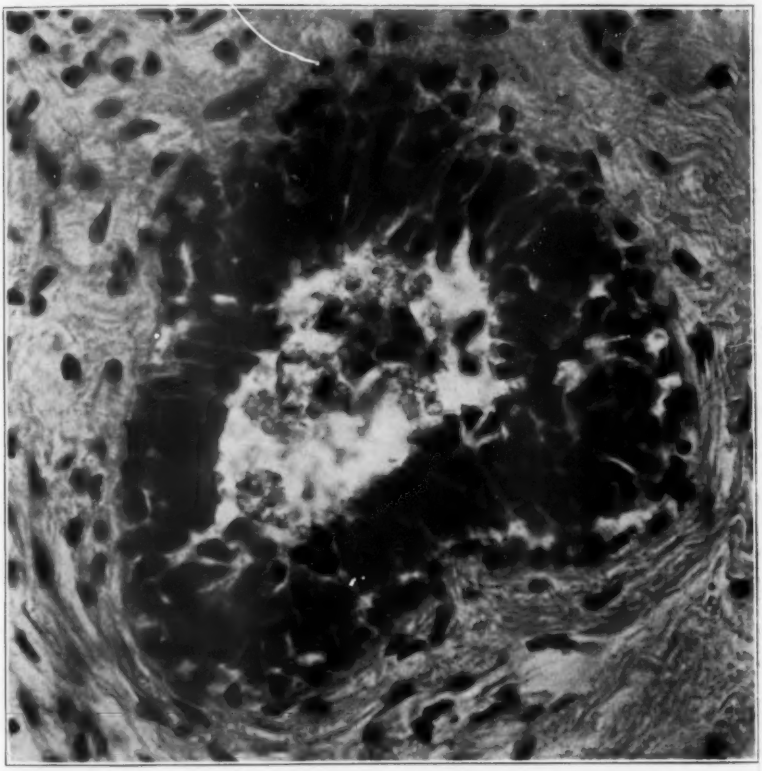

Fig. 6 (path. no. 36144).—Microscopic structure of breast tissue from a colored man, 27 years of age, who, for nine months, complained of a gradual enlargement of his breast associated with sharp, radiating pains. The mass was excised, and the patient is living seven years later. The microscopic section shows an epithelial hyperplasia, with the cells varying from oval to cuboidal in shape. Many of these cells have desquamated into the duct lumen. The periductal stroma is composed of adult fibrous tissue.

Physical examination gave essentially unimportant results except for a flat, firm, slightly tender, freely movable, lobulated tumor, underlying the nipple and located in the upper and outer quadrant of the left breast. After a diagnosis of a benign lesion, a biopsy was decided on.

Fig. 7 (path. no. 23500).—Microscopic structure of breast tissue from a colored man, 33 years of age, who complained of a slowly growing nodular mass in his left breast which was painful at times. The duration was three years. The mass was excised, and the patient had an uneventful recovery. The microscopic section shows a typical intracystic papilloma in a case of gynecomastia. Since different stages of papilloma formation have been noted in gynecomastia (fig. 3), papilloma may be considered one of the later stages of that condition.

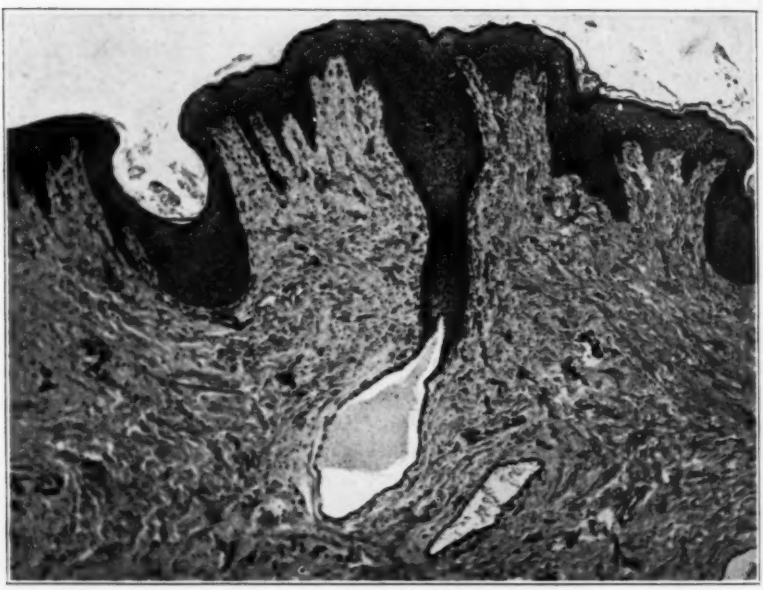

Fig. 8 (path. no. 12265).—Microscopic structure of breast tissue from the nipple of the normal breast of a white man, 35 years of age, showing a dilatation of the mammary duct.

The mass was excised and a frozen section proved it to be a benign lesion.

Microscopic examination showed hyperplasia of the duct epithelium with a slight desquamation into the lumen (fig. 9). The periductal stroma was composed of dense, adult fibrous tissue with no evidence of recent hyperplasia. There was no marked periductal infiltration of wandering cells.

The patient made an uneventful recovery, but the ultimate result is not known for all trace of the patient is lost thirteen years later.

The comparison of the foregoing case with the microscopic section (fig. 10) of a 21 year old woman who suffered from a slightly painful

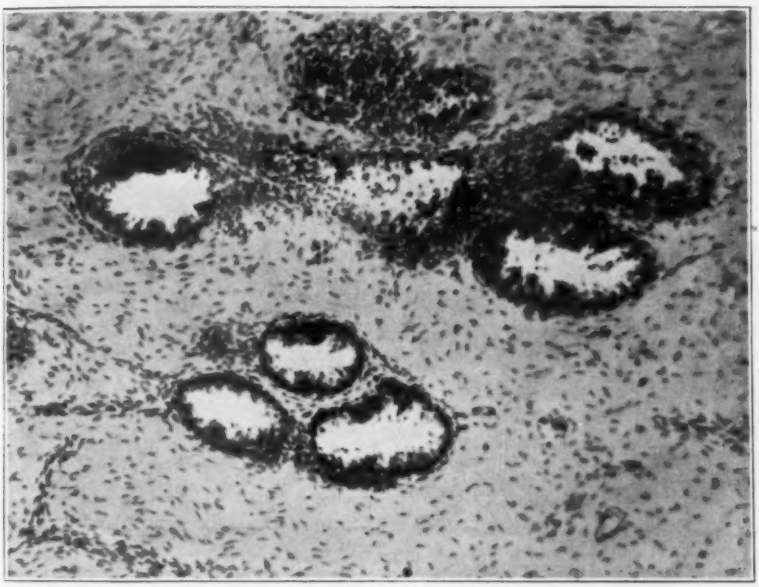

Fig. 9 (path. no. 23500).—Microscopic structure of breast tissue from a colored man, 33 years of age (fig. 8). The microscopic section shows epithelial hyperplasia with desquamation into the duct lumens. The periductal fibrous tissue is dense, collagenous and indicates hypertrophy of long duration.

nodule in her breast for one year shows the close relationship existing between an apparent hypertrophy of a male breast and fibro-adenoma of a female breast.

These studies seem to indicate that hyperplasia grades directly into fibro-adenoma with no definite line of demarcation. Because of this imperceptible gradation, it is impossible to classify borderline cases definitely, and hence the fibro-adenomatous hypertrophy is classed as a further extension of hyperplasia.

SWEAT GLANDS

The intimate relationship of the mammary glands to sweat glands has been proved by Scheifferdecker.[84] The microscopic resemblance of the mammary ducts to the tubules of the sweat glands is so close in some cases that it is difficult to distinguish between them. For practical purposes, the chief differentiating point is the presence or absence of desquamation.

The breast, being a holocrine gland, contains desquamating cells in the lumen of its ducts, while the sweat gland, being a merocrene gland,

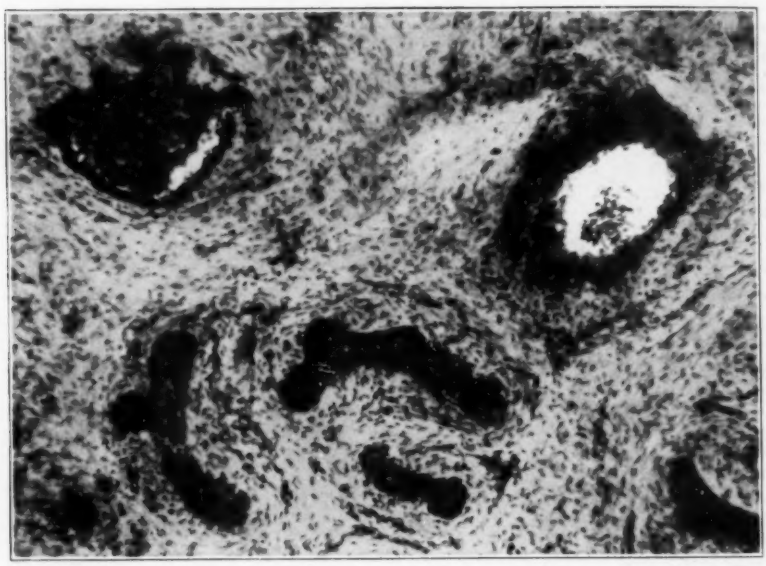

Fig. 10 (path. no. 2250).—Microscopic structure of the breast tissue from a white woman, 21 years of age, who, for one year, complained of a small painful nodule in her breast. The mass was excised, and the patient is well five years later. The microscopic section reveals an epithelial hyperplasia with desquamation into the duct lumen, embedded in a dense adult fibrous stroma. Compare this fibro-adenoma with the hypertrophy shown in figure 9.

secretes a clear eosin staining material (fig. 11). The secretion of the large sweat glands, which are located in the breast, are believed by Loeschcke[85] and Scheifferdecker[84] to carry volatile substances which stimulate sex lure. Von Gusnar,[16] in studying the histology of the male

84. Scheifferdecker: Zoologica, 1922, no. 72.

85. Loeschcke, H.: Ueber zyklische Vorgänge in den Drüsen des Achselhöhlenorgans und ihre Abhängigkeit vom Sexualzyklus des Weibes, Virchows Arch. f. path. Anat. **255**:283, 1925.

breast, demonstrated the different forms of sweat glands resembling the mammae. He also pointed out adenomas of the sweat gland and cystic dilatations occurring in the normal breast which are frequently mistaken for mammary parenchyma.

GROSS DESCRIPTION

Since gynecomastia comprises such a large field, the gross pathology does not present a typical picture. Specimens collected show encapsu-

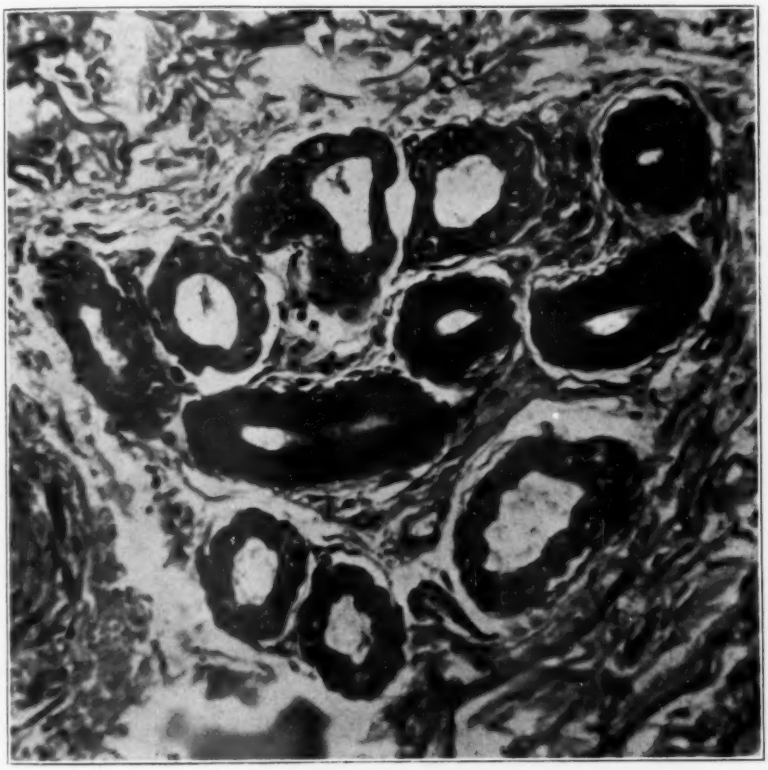

Fig. 11 (path. no. 44224).—Microscopic structure of sweat glands from a white man, 23 years of age (fig. 12), with gynecomastia. These merocrene glands are frequently mistaken for parenchyma of the breast. They are characterized by a clear, nondesquamated, eosin-staining secretion in their lumens. They are divided into two types, namely, the apokrine (large) and the ekkrine (small) sweat glands. The ekkrine glands are distinguished by their darker staining qualities.

lated, localized or discrete, and diffuse growths. However, in general, the prevailing tissue is fibrous with scattered islands of semitranslucent parenchyma. All the hypertrophies are apparently a variation of this fundamental picture with the supportive element predominating.

The gross specimen (fig. 12 *A* and *B*) usually consists of a uniform, circumscribed mass varying from 2 cm. to 4 inches in diameter. It is usually white, bluish-white or gray and is surrounded by fat, and sometimes by a thin fibrous capsule. It may be attached to the skin, but

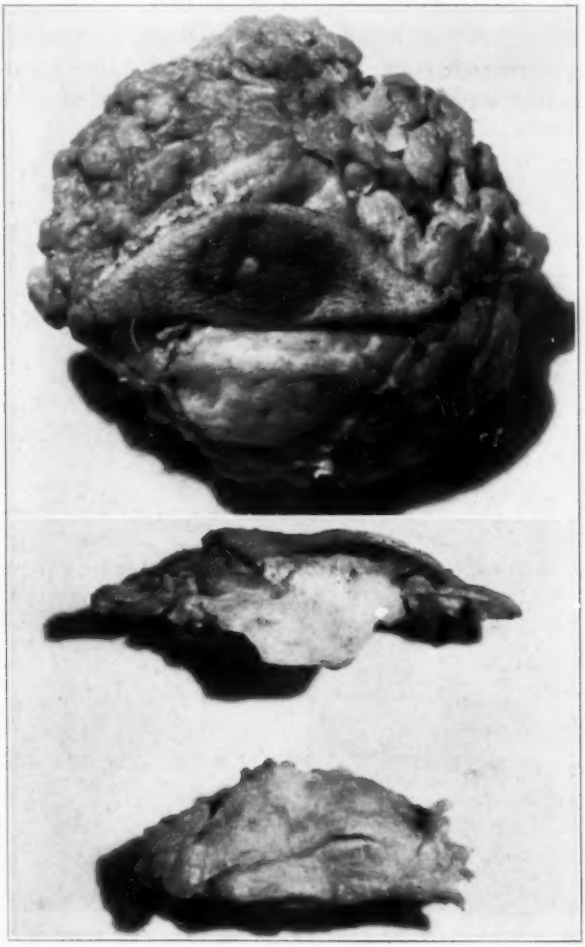

Fig. 12 (path. no. 44224).—Specimen from a white man, 23 years of age, with a history of tenderness and a small nodule posterior to the left nipple. Four years after the onset, the mass was excised. The microscopic section confirmed the diagnosis of gynecomastia. The patient is living and well one year later. The gross specimen reveals a circumscribed mass posterior and inferior to the nipple which, on section, shows dense fibrous striations infiltrated with bluish-white tissue and dotted with occasional yellow opacities.

never to the underlying muscles. The consistency may be hard, firm, rubbery or soft.

The mass is easily sectioned, and on detailed examination it usually reveals a homogeneous, white, bluish-white or gray surface which may be dotted with pink or grayish, translucent areas projecting between the strands of fibrous tissue. Yellow opacities may also be found on the cut surfaces.

The acute and diffuse hypertrophies usually have a white or bluish-white translucent appearance, while the chronic and localized forms usually present a white, gray, opaque surface.

DIAGNOSIS

All swellings of the male breast are not produced by hyperplasias. This fact is evident from an examination of the present series of benign tumors of the male breast which includes dermoid cyst, lipoma, lymphangioma and the specific infections of tuberculosis and syphilis, together with nonspecific infections.

From the statistics already given (see tabulation), gynecomastia is more prevalent in the white race; it occurs more frequently in persons between 31 and 40 years of age (fig. 13); it has an average duration of 14.7 months; it occurs equally on the right and left sides; it is bilateral in 12.8 per cent of the cases; it may recur; it is not usually associated with trauma or pain (fig. 14); it is associated with tenderness in 50 per cent of the cases; the tumor is usually single, and is multiple in only 6.2 per cent of the cases. The consistency varies from soft and moderately firm to rubbery and hard.

The recognition of diffuse hypertrophies, both unilateral (fig. 15) and bilateral, is comparatively easy. However, the presence of a discrete nodule presents a more complex picture (fig. 16). A history of fluctuation in size, a multiplicity of lesions and a bilateral occurrence are in favor of a benign lesion.

In spite of the fact that malignancy is evident neither in the microscopic picture nor in our follow-up reports, such a development may occur. Berns [85] reported that a cancer developed in one of two enlarged mammary glands of a man, and that it was the ultimate cause of death. He stated that the breasts were similar to those of a woman and that the carcinoma recurred after an amputation. Among his case reports on gynecomastia, Erdheim [57] included a malignant intraduct papilloma. Twenty-three per cent of the tumors of the male breast are malignant; therefore, whenever a nodule is palpated, malignancy should be definitely ruled out or confirmed. If the mass shows no tendency to fluctuate in size, if it continues to grow or if it presents an indefinite picture, a biopsy and frozen section of it should be made.

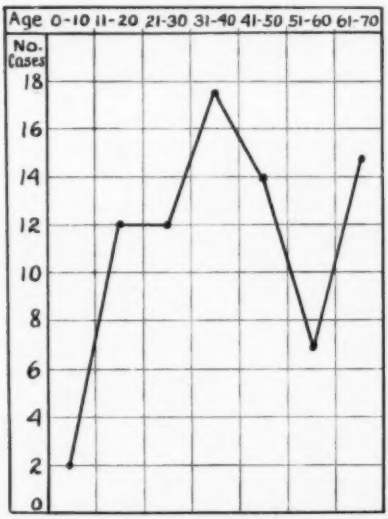

Fig. 13.—Chart of the age incidence in eighty cases of gynecomastia.

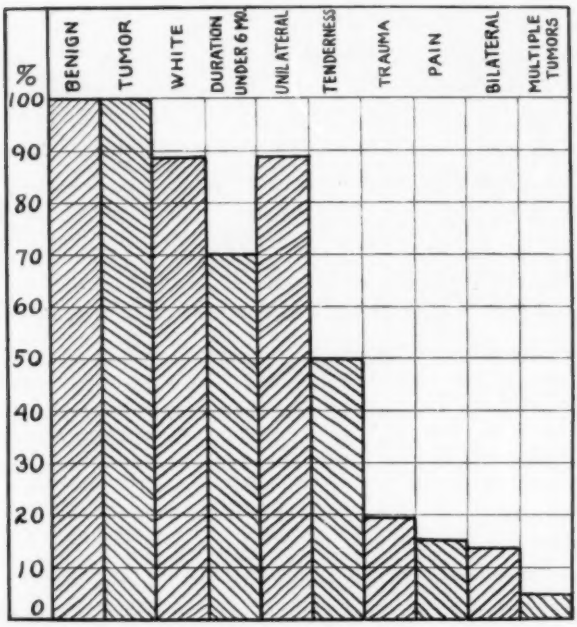

Fig. 14.—Chart indicating the percentages of the outstanding signs and symptoms in the present series of eighty-eight cases of gynecomastia.

TREATMENT

In view of the findings on roentgen treatment recently reported,[86] it should be given a trial in all cases of acute hypertrophy. Evidence leads to the belief that chronic hypertrophies are unaffected by irradiation therapy.

Diffuse enlargements without nodular formation which do not respond to irradiation should be let alone unless the patient insists

Fig. 15 (path. no. 32768).—Diffuse symmetrical enlargement of the right breast in a white boy, 12 years of age. There was a history of gradual, diffuse, painful swelling of six months' duration. Three deep roentgen treatments were administered with a gradual and, finally, a complete disappearance of the tumefaction four months after the last treatment. The patient is living and well, with no recurrence eight and a half years after the last treatment.

on removal for cosmetic purposes. In such cases, plastic surgery may be resorted to. As a rule, the nipple is of little consequence to the male patient and in such cases an elliptic excision, including the nipple, areola and breast tissue, may be made. However, when the breast is greatly

86. Menville, John G.: X-Ray Treatment in Gynecomastia, Radiology, to be published.

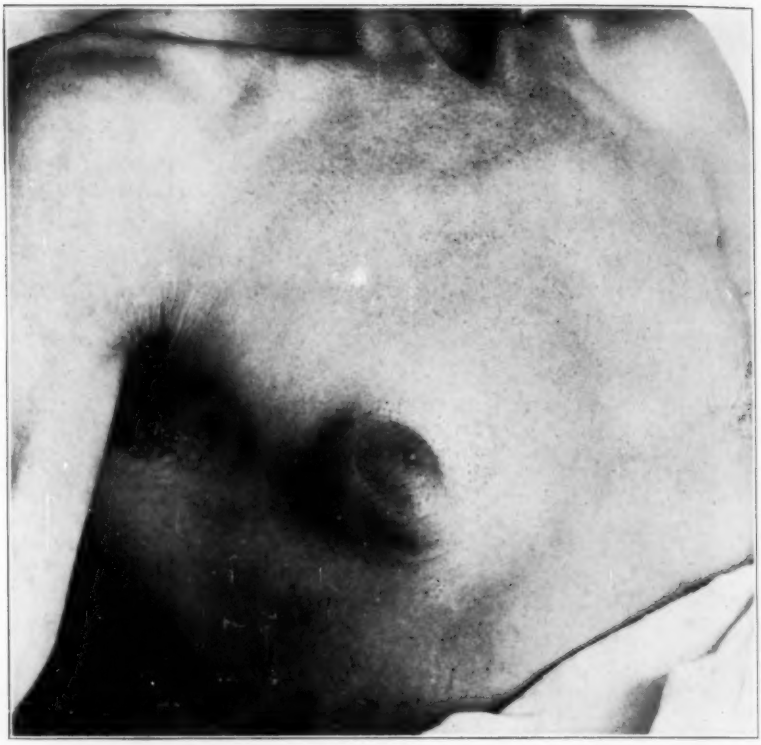

Fig. 16 (path. no. 24617).—Circumscribed discrete swelling posterior and lateral to the right nipple in a white man, 48 years of age. There was a history of a gradually growing nodular mass posterior to the nipple of three years' duration. The breast was excised, and the patient is well eight years later.

Fig. 17.—View of a whole male breast section showing the appearance of gynecomastia at very low magnification. This breast was removed because malignancy was feared. High power magnification verified the diagnosis of gynecomastia.

enlarged and the patient does not care to lose the nipple, the operator may utilize a thoracolateral incision at the junction of the edge of the breast and thoracic wall. This latter incision saves the nipple, leaves a minimal scar and permits the removal of breast tissue. Pain, which may be associated with diffuse hypertrophies, may be treated by suggestion, although if it proves to be a constant source of physical and mental anxiety excision should be performed.

A good rule to follow in hypertrophies which are either composed of, or include, a discrete nodule is to do a biopsy and make a frozen section in order to rule out malignancy. The biopsy performed in cases of localized hypertrophy should constitute a total excision of the tumor mass. Such a procedure answers the purposes of both diagnosis and treatment.

PROGNOSIS

The present series reveals no evidence of malignant change in 88 cases of gynecomastia. However, the isolated reports in the literature of such cases serve as a warning that this possibility should always be remembered. The prognosis concerning the deformity is good if the proper treatment is instituted.

CONCLUSIONS

1. Gynecomastia results from general and local factors.

2. A study of 130 cases of tumors of the male breast revealed:

(*a*) The hypertrophies occurred in 88.8 per cent of the total cases.

(*b*) The so-called fibro-adenoma is a further development of hypertrophy of the male breast (gynecomastia).

(*c*) Fibro-adenoma is included in the classification of gynecomastia.

3. All doubtful cases presenting a discrete nodule should have the benefit of a biopsy and a frozen section to rule out malignancy.

DUODENAL ILEUS

EFFECT OF CHRONIC DUODENAL OBSTRUCTION ON EVACUATION OF THE GALLBLADDER

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AND
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One of the phases of chronic duodenal ileus which has attracted particular attention is the relation between it and the function of the biliary tract. Every clinical description of duodenal ileus includes, as one of its frequent manifestations, a symptom complex which simulates disease of the gallbladder.[1] Epigastric distress coming on shortly after meals, pain in the right hypochondrium with scapular radiation, eructation and even jaundice have been described either with or without organic changes in the biliary tract.[2] Orndoff [3] suggested a relationship between the disturbances of duodenal motility so frequently seen in pregnancy and the cholecystic symptoms and disease which may attend and follow it. Duodenal ileus is said in some way to render the biliary tract more liable to infection.[4] Wheelon [5] found an involvement of the biliary structures in 75 per cent of seventy-four patients with duodenal ileus. A cholecystitis had developed in thirty-three cases, hepatitis in fourteen and peribiliary adhesions in forty-eight. In the remaining cases the biliary structures were unchanged.

From the Surgical Pathological Laboratory of the College of Physicians and Surgeons, Columbia University, and the Departments of Surgery and Roentgenology of the Presbyterian Hospital.

1. Andrews, K. S.: Chronic Duodenal Ileus, M. Clin. North America **13**:1027, 1930. Wilkie, D. P. D.: Chronic Duodenal Ileus, Am. J. M. Sc. **173**:643, 1927.

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3. Orndoff, B. H.: Reverse Movements in the Contents of the Duodenum and Its Probable Significance, Illinois M. J. **55**:406, 1929.

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5. Wheelon, Homer: Symptoms Associated with Duodenal Retention and Reverse Motility, J. A. M. A. **86**:326 (Jan. 30) 1926

Physiologic interest has centered on the much disputed rôle of the duodenum in the control of biliary outflow. One or more, or all, of the factors proposed, such as contraction of the musculature of the gallbladder, elastic recoil of the wall of the gallbladder, biliary secretory pressure with suction action on the gallbladder contents by the common bile duct stream flowing past the cystic duct and the changes in the intra-abdominal pressure with respiratory movements, empty the glallbladder and carry bile to the duodenum.[6] Because of the long and oblique insertion of the common bile duct in the substance of the duodenal musculature, the waves of duodenal peristalsis sweeping downward are said to exert an important milking action on biliary flow.[7] Aspiration, by the reduced pressure in the duodenum which follows each peristaltic wave, serves to complete the discharge of bile.[8] Resistance to the outflow of bile is controlled by tonus changes in the duodenal wall,[9] by a special sphincter at the orifice of the duct,[10] or perhaps by both.[11]

In the following experiments, the attempt was made to reproduce artificially the conditions of duodenal ileus, to alter duodenal tonus and motility and to study the changes which this might produce in the evacuation mechanism of the gallbladder.

METHOD

In the course of certain studies on the etiology of gallstones, Walsh and Ivy,[12] in an attempt to obtain stasis of bile through a permanent alteration in duodenal motility, employed a method of duodenal reversal. A 2 inch (5 cm.) segment of

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7. Carlson, A. J.: Physiology of the Liver: Present Status of Our Knowledge, J. A. M. A. **85**:1468 (Nov. 7) 1925. Copher, G. H.; Kodama, S., and Graham, E. A.: The Filling and Emptying of the Gall Bladder, J. Exper. Med. **44**:65, 1926.

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12. Walsh, E. L., and Ivy, A. C.: Observations on the Etiology of Gall Stones, Ann. Int. Med. **4**:134, 1930.

the third portion of the duodenum was excised from duodenal continuity without disturbing its mesentery or blood supply. It was then turned 180 degrees on its transverse axis and sutured at both ends to reestablish duodenal continuity. Its distal end, however, was then directed orally; its proximal end, aborally. The normal peristaltic activity of the reversed segment would thus be directed up-stream, opposed to the peristaltic drive from above, producing in effect a physiologic duodenal obstruction. In the course of eight months, this led to certain morphologic and chemical evidences of biliary stasis in Walsh and Ivy's cases. There was a marked hyperplasia of the mucosa and lymphoid tissue of the gallbladder. At autopsy the bile was found to be thick, and it contained sediment of the pigment and carbonate type. To reproduce duodenal ileus we employed, at first, this reversal method.

Rather small dogs (from 8 to 12 Kg.) were used generally to facilitate handling under the x-ray machine and fluoroscope. Control studies of the gastric and duodenal activity were conducted, and control emptying times of the gallbladder and stomach were determined before operation. The method of combined visualization, suggested by Levene and Whitaker,[13] clinically, was found to be useful and reliable in the dog. In general, for visualization of the gallbladder, the technic described by Boyden,[14] Graham [15] and Copher [16] was used. No food was allowed on the day of the injection of the dye. Tetiothalein sodium (Mallinckrodt) in a single dose of 0.15 Gm. per kilogram of body weight, dissolved in 30 cc. of water with 2 drops of 2 per cent sodium hydroxide and filtered through China silk, was sterilized in the autoclave. It was injected intravenously at 5 p. m. Thereafter, water also was withheld. At 8 a. m. the next morning, fifteen hours later, roentgenograms were taken. When good visualization of the gallbladder was determined, a combined motor and gallbladder emptying meal was given. The meal consisted of 120 cc. of 20 per cent cream, two egg yolks, 16 Gm. of barium sulphate and enough water to make a total bulk of 240 cc. Fluoroscopy was conducted, and roentgenograms were taken at sufficiently frequent intervals to determine the time required for emptying of the gallbladder and for emptying of the stomach, and to make observations on duodenal motility. This simultaneous study of gastroduodenal and gallbladder activity admits of closer correlation of the results obtained, but the readings made on separate study of each activity were just about the same.

Generally, under the conditions described, the gallbladder emptied in from one and one-half to two hours. In one dog it took three hours, and in two others it

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13. Levene, G., and Whitaker, L. R.: New Methods for Clinical Study of Gall-Bladder: Elaboration of Cholecystography with Simultaneous Examination of Stomach and Duodenum, New England J. Med. **202**:203, 1930.

14. Boyden, E. A., and Birch, C. L.: Reaction of the Gall Bladder to Stimulation of the Gastro-Intestinal Tract: Response to Substances Injected into Duodenum, Am. J. Physiol. **92**:287, 1930. Boyden, E. A.: Effect of Natural Foods on Distention of the Gall Bladder, Anat. Rec. **30**:333, 1925.

15. Graham, E. A.; Cole, W. H., and Copher, G. H.: Cholecystography: An Experimental and Clinical Study, J. A. M. A. **84**:14 (Jan. 3) 1925; Cholecystography: The Use of Sodium Tetraiodophenolphthalein, ibid. **84**:1175 (April 18) 1925. Graham, E. A.: Diseases of the Gall Bladder and Bile Ducts, Philadelphia, Lea & Febiger, 1928.

16. Copher, G. H.: Cholecystography: Appearance and Disappearance of the Shadow, J. A. M. A. **84**:1563 (May 23) 1925.

took five hours. No reason could be found for these normal departures from the average, and repetition of the experiments confirmed within from one-half to one hour the normal control emptying time of these dogs. The stomach emptied itself in from four to five hours. In only one dog did it take six hours. The range of normal gastric evacuation time was found to be somewhat greater than that of the gallbladder, viz., from one to two hours. Before operation no duodenal stasis, dilatation or antiperistalsis was ever observed. Duodenal peristalsis was uniform, rhythmic and infrequent.

RESULTS

The effort to produce a chronic duodenal ileus by the reversal method sufficiently marked to give definite fluoroscopic changes was unsuccessful in our hands. Just how long a segment of the small intestine needs to reverse to cause the required amount of physiologic obstruction without killing the animal by too great an obstruction had to be determined by trial and error. At first a 3 cm. segment of the most distal portion of the duodenum was used. At the first determinations taken ten days after operation, the gastric and cholecystic emptying times were exactly the same as before operation. There was no apparent increase in duodenal activity, antiperistalsis, dilatation or stasis. Then a 6 cm. segment of the same portion of duodenum was used, with still no apparent results. Since the reversal of longer duodenal segments would have encroached on the region of the ampulla, it was decided that for longer reversal segments the first loop of jejunum would be used. It was recognized that probably longer segments of jejunum than of duodenum would be required to give the same amount of obstruction. A 15 cm. segment of jejunum was therefore employed. The results were the same—negative. There was what might have been interpreted fluoroscopically as a slight hyperactivity of the duodenum. Autopsy showed a slight hypertrophy of the duodenum, but there were no definite physiologic changes sufficient for the purposes of our experiments.

As a last resort, we reversed a 40 cm. portion of small intestine, beginning proximately quite close to the ligament of Treitz (dog 584). In this small dog the segment represented a considerable portion of the entire small intestine. Ten days after operation, fluoroscopy revealed the duodenum to be fully twice its preoperative diameter. The gastric emptying time was increased by three and one-half hours. The meal remained in the dilated duodenum for fully one hour after the stomach was empty. Peristaltic activity of the duodenum was markedly exaggerated. Wave after wave passed hurriedly down the dilated duodenum in quick succession, with little or no intervals of comparative quiet. The waves would pass down to the reversed segment, stop and charge backward toward the pylorus. Peristalsis and antiperistalsis kept chyme surging rapidly back and forth several times, before a thin stream was

admitted into and beyond the reversed segment. In the reversed jejunal segment activity was increased, but not as markedly as in the duodenum. Chyme was seen to pass in it alternately in both directions, up toward the stomach and down toward the ileum. Beyond the reversed segment chyme passed only distally and quietly. The evacuation time of the gallbladder was markedly increased. Before operation, it had taken only two hours; after operation, the gallbladder required fifteen hours to empty itself completely.

Thirty days after operation there was little change from the picture just described. The evacuation time of the gallbladder was still fifteen hours and the gastric evacuation time was fifteen hours instead of the preoperative four hours. Duodenal hyperactivity, however, was not quite so marked as at the ten day observations, and the dilatation had somewhat subsided. Fifty days after operation everything returned to normal. There was still a slight hyperactivity, but no dilatation, stasis or delay in the emptying of the stomach or gallbladder. Autopsy revealed the reversed segment moderately hypertrophied and the duodenum proximal to it markedly hypertrophied, with no gross changes in the biliary tract or the bile. Whatever the resulting morphologic changes may have been, complete physiologic compensation for the reversal had apparently occurred.

Reversal of a 40 cm. segment of jejunum was tried on a second dog. Even the first observations, taken seventeen days after operation, gave results exactly similar to those before operation. Because of the variability of results and the dog's unexpected facility to compensate even for such large reversals, this method was finally abandoned. It may be that the reversal of still longer segments would finally have given constant and permanent results. However, it mattered little for the purposes of these experiments how the duodenal ileus was produced, and it was decided at this point to change to a more certain method.

Berg and Jobling [17] have described a method of producing chronic duodenal obstruction which gives fairly constant results. An upper right paramedian incision is made; a flap about 4 cm. wide and 4 cm. deep, with its base lateral, is cut in the lateral edge of the posterior sheath. An opening is made in an avascular field of the duodenal mesentery just proximal to the ligament of Treitz. The flap is passed from right to left through this opening and turned back laterally over

17. Berg, B. N.; Meleney, F. L., and Jobling, J. W.: Experimental Chronic Duodenal Obstruction: I. Technic and Physiology, Arch. Surg. **14**:752 (March) 1927. Berg, B. N., and Jobling, J. W.: Experimental Chronic Duodenal Obstruction: Changes in the Blood and Other Pathologic Changes, ibid. **16**:593 (Feb.) 1928.

the duodenum, forming for it a sort of sling or ventral mesentery. The edge of the reflected flap is sutured closely to its own base, by four or five interrupted mattress sutures, in such fashion that a marked constriction of the lumen results. In general, a flap the width of which is one and one-half times the diameter of the duodenum (roughly one-half its circumference) gives, when formed into the sling, an effective degree of constriction without jeopardizing the circulation of the bowel. This is not altogether constant, for both the sling and the wall of the bowel are slightly variably plastic. A few animals were lost because of excessive obstruction or because the sling had ulcerated through, and operations had to be performed a second time on a few animals to take

Fig. 1 (dog 408).—Ten days after operation; two hours after the ingestion of a barium meal. Note the sling constriction in the third portion of the duodenum, with the bulblike dilatation just proximal to it and the moderate dilatation of the rest of the proximal duodenum.

up the slack in the sling and obtain a more effective ileus, but in general the method gave satisfactory results. This method has the advantage over compression by metallic bands in that no foreign body is involved and the sling suspends the duodenum to the ventral abdominal wall so that fluoroscopic visualization is facilitated.

Chronic duodenal obstruction was finally obtained by this method. In eight dogs an ileus was produced which was even more striking than the one positive result described in the reversal method. In all eight dogs, ten days after operation, there were duodenal hyperactivity, anti-

peristalsis, dilatation and stasis. The gastric emptying time was usually, but not always, increased, by from one and one-half to five hours. The contrast between the duodenum proximal to the sling and the jejunum just beyond it was striking (fig. 1). The duodenum in some cases appeared fluoroscopically to be three times its preoperative width. Waves of turbulent peristalsis and antiperistalsis carried chyme quickly back and forth in the duodenum, and only an occasional fine trickle was admitted beyond the obstruction. In comparison with the fluoroscopic findings, the symptoms presented by these dogs were slight. An equivalent degree of duodenal dilatation and hyperactivity in human beings would have been accompanied by attacks of biliary vomiting leading toward tetany and to a marked loss of weight and strength. In these dogs, unless the obstruction soon proved incompatible with life, vomiting occurred only rarely and, if at all, only in the first two postoperative weeks. The animals were kept on the ordinary stock diet, except that bones were prohibited and an extra ration of chopped meat was allowed. They lost weight at first, but after a time regained most of it. There was no anorexia or asthenia despite the widely dilated and hyperactive duodenum.

Duodenal ileus resulted in each case, but the degree of change in each manifestation of the ileus, especially in their relation to one another, varied greatly. Berg and Jobling found the same lack of correlation. Sometimes there was marked hyperactivity with slight dilatation, sometimes slight hyperactivity with a widely dilated duodenum and all variations between. The same variability in relation was noted between duodenal hyperactivity and duodenal stasis, between the dilatation and stasis and between any or all of these conditions with gastric stasis. In two dogs (nos. 408 and 645) no gastric stasis resulted despite the marked duodenal obstruction.

The ileus obtained was permanent in all but one dog. The observations taken thirty and sixty days after operation, and even later, showed essentially the same ileus observed at ten days. There was again, however, a lack of correlation between its individual signs. There was, furthermore, a great variability with each manifestation between its intensity at any one time and that at previous or later readings. Yet the obstruction, in one or more of its variable features, persisted. At subsequent autopsies on these animals, the obstructed duodenums were found to be abruptly wider than the jejunums by from 40 to 80 per cent. The duodenal musculature above the obstruction was hypertrophied to almost twice the thickness of the duodenal muscle below the obstruction. When it is considered that the duodenum was at the same time dilated, the actual hypertrophy is seen to be even greater than the increase in thickness implies. There was no duodenitis, neither was there

evidence of any gross morphologic change in the biliary tract (see also the histologic studies of Berg and Jobling). In only one dog (no. 586) did all manifestations of the ileus, including hyperperistalsis, antiperistalsis, dilatation and stasis, subside. In this animal only a slight obstruction was found at autopsy. The sling admitted fully the tip of the little finger, although at operation it had been left much tighter. The duo-

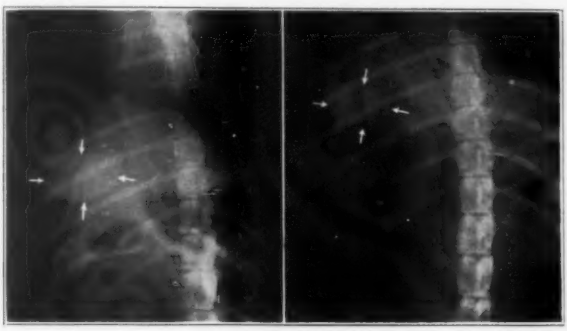

Fig. 2 (dog 598).—Preoperative study of evacuation of the gallbladder. The film on the left shows the gallbladder visualization before the ingestion of a cream-egg yolk meal. The film on the right, taken three hours afterward, shows the gallbladder shadow fainter and smaller. At four and one-half hours only a faint trace of dye remained. At five and one-half hours the gallbladder shadow was absent.

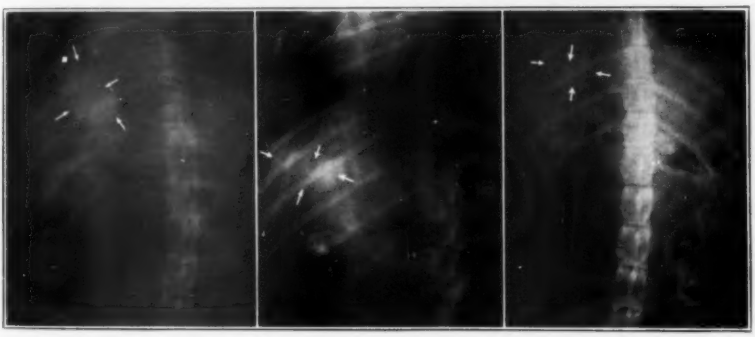

Fig. 3 (dog 598).—One month after operation (sling). The film on the left shows the gallbladder visualization before the ingestion of a cream-egg yolk meal. The film in the middle, taken eight hours afterward, shows the gallbladder of the same size and the same dye intensity. The film on the right, taken fourteen hours later, shows a moderate-sized gallbladder shadow. At eighteen hours only a faint trace of dye remained. At twenty-two hours the gallbladder shadow was absent.

denum above it was also hypertrophied, indicating that at some recent time, at least, a greater obstruction had been present. Stretching of the sling together with a simple increase in the force of peristaltic con-

traction may have been responsible for the gradual subsidence of ileus in this one dog.

In all of the nine dogs in which duodenal ileus had been produced, there was found at first a definite decrease in the efficiency of evacuation of the gallbladder. Ten days after operation, in response to the same emptying meal, the evacuation time of the gallbladder was increased above its preoperative limits. This increase varied anywhere from one to ten hours. Here, too, the degree of increase could not be correlated with all or any one of the manifestations of ileus. Only one conclusion could be derived from these readings; i. e., in all nine dogs the primary result of duodenal ileus was invariably a definite delay in the emptying of the gallbladder (figs. 2, 3 and 4). During the unsuccessful attempts to produce duodenal ileus either by the reversal or by the sling method,

Fig. 4 (dog 584).—One month after operation (reversal). The film on the left shows fasting gallbladder visualization. The film on the right, taken thirteen hours after the ingestion of a combined cream-egg yolk-barium meal, shows a small gallbladder shadow still present, barium in the ileum and colon and a small gastric residue. At sixteen hours the stomach was empty and the gallbladder shadow was absent.

none of the dogs (six) ever showed an increase in the evacuation time of the gallbladder. These dogs served as controls to prove that the ileus and not the operation was responsible for the delay in the emptying of the gallbladder.

In seven of the nine dogs observations on the emptying time of the gallbladder were obtained thirty and sixty days after operation, and even later. In two of these dogs (nos. 342 and 667), in the course of time, the duodenal ileus increased and the delay in evacuation of the gallbladder increased. In two dogs (no. 586, with the sling, and no. 584, with the reversal) all signs of ileus subsided, and with it the evacuation time of the gallbladder returned to normal. In the remaining three dogs

(nos. 598, 619 and 408) the duodenal ileus remained, and yet the emptying time of the gallbladder returned to normal (table).

One incidental experience should be mentioned. Many of the preoperative control animals had to be discarded because of the onset of distemper. It was noted that if the animals were coming down with distemper, even a few days before frank "snuffles" and diarrhea had begun, the biliary flow was sluggish. The evacuation time of the gallbladder in these control animals increased to fifteen, twenty or more hours. We learned to recognize this sign of biliary stasis as a warning

Results

Dog	Condition	Gallbladder, Emptying Time in Hours	Stomach, Emptying Time in Hours	Duodenal		
				Hypermotility	Dilatation	Stasis
442	Before operation	2	4½	0	0	0
	After operation, 10 days	5	7	4+	3+	2+
645	Before operation	1½	4½	0	0	0
	After operation, 10 days	6	4½	4+	4+	2+
342	Before operation	5	5	0	0	0
	After operation, 10 days	6	7	4+	1+	0
	After operation, 30 days	8½	4½	4+	3+	2+
667	Before operation	1½	5	0	0	0
	After operation, 10 days	4	6½	2+	2+	1+
	After operation, 40 days	7	10	3+	4+	4+
586	Before operation	2	4	0	0	0
	After operation, 10 days	9	9	4+	2+	2+
	After operation, 40 days	4	9	2+	2+	2+
	After operation, 60 days	2½	6	1+	1+	0
584	Before operation	2	4½	0	0	0
	After operation, 10 days	15	7½	4+	2+	2+
	After operation, 30 days	15	15	3+	1+	2+
	After operation, 50 days	2	4	1+	0	0
598	Before operation	5	5	0	0	0
	After operation, 10 days	15	9	4+	2+	2+
	After operation, 30 days	20	5	4+	2+	2+
	After operation, 60 days	6	4½	2+	2+	1+
	After operation, 90 days	3½	5½	4+	1+	1+
619	Before operation	3	5	0	0	0
	After operation, 10 days	7	7	4+	2+	2+
	After operation, 30 days	5	3½	4+	0	0
	After operation, 60 days	3½	4	3+	2+	3+
408	Before operation	2	6	0	0	0
	After operation, 10 days	3	6	1+	4+	3+
	After operation, 30 days	6	9	2+	4+	3+
	After operation, 60 days	2½	12	2+	4+	3+

that distemper was beginning, and avoided further use of the animals. Because of the biliary stasis with infection, when distemper developed in any of the animals operated on, further results on them were dropped as unreliable.

COMMENT

According to Alvarez' gradient theory,[18] any given point of intestine is more highly irritable than the next point distal to it, and normal peristalsis, therefore, proceeds only aborally. Antiperistalsis can appear

18. Alvarez, W. C.: The Mechanics of the Digestive Tract, New York, Paul B. Hoeber, Inc., 1928. Cannon, W. B.: The Mechanical Factors of Digestion, New York, Longmans, Green & Co., 1911.

under conditions of obstruction or local irritation, but otherwise the proximal-distal polarity of the intestine is constant. It is generally believed that this polarity of the peristaltic gradient is irreversible. The surgical reversal of a segment of intestine should therefore direct its peristaltic waves permanently up-stream against the downward drive of the proximal parts of the intestine, and thus produce a definite physiologic obstruction. Yet after each reversal complete compensation occurred, and no permanent ileus was produced.

The failure of the reversal method to produce chronic duodenal ileus may be accounted for by:

1. An exaggerated activity of the intestine proximal to the reversed segment of such force that chyme is propelled through and beyond the reversed segment in spite of the physiologic obstruction which it imposes.

2. An increase in antiperistalsis in the reversed segment proper. This would carry chyme down-stream against the upward drive of normal peristalsis in the anatomically reversed segment.

3. A reversal of physiologic polarity in the anatomically reversed segment. This would direct the peristalsis down-stream again and relieve the obstruction.

That there was no reversal of polarity is suggested by the observation that within the reversed segment chyme was seen to pass alternately in both directions. It was carried distalward either by propulsion by the duodenum proximal to the reversed segment or by antiperistalsis within the reversed segment. It could be carried in the proximal direction, however, only if the normal up-stream directed peristalsis of the reversed segment persisted, i. e., only if polarity was not reversed. This suggestion is supported by the moderate hypertrophy of the muscularis of the reversed segment. If reversal of polarity had occurred, the reversed segment would not be driving chyme up-stream, and hypertrophy would not have taken place. The marked hypertrophy of the proximal duodenum attested further to the persistent resistance to the aboral passage of chyme in the reversed segment, which had to be and was successfully overcome by an increased motor drive of the intestine proximal to it.

The duodenal ileus obtained by the sling method, by reason of its anatomic character, was practically constant. It was also fairly uniform except for slight unavoidable differences in the formation of the sling. It, too, however, elicited all the means of compensation which could be applied. Peristaltic activity and muscular tonus of the duodenum proximal to the sling increased. With a given obstruction, if the tonus increases markedly, there is little dilatation; if only slightly, there is more dilatation.[19] With a given increase of tonus the greater the obstruction the

19. Koennecke, W., and Meyer, H.: Klinisches und Experimentelles zur chronischen Duodenalstenose, Deutsche Ztschr. f. Chir. **175**:179, 1922.

wider will be the dilatation. Stasis results only when the obstruction is greater than peristaltic efficiency can easily overcome.[20] As long as gastric peristaltic efficiency can meet its increased load there is no gastric stasis. If the duodenum can similarly meet its increased load, there is no duodenal stasis. Antiperistalsis appears because of the local exaggerated irritability. But in spite of the counter, up-stream drive, peristalsis and tone can be so elevated that no stasis results. However, peristaltic reaction and tone vary greatly even normally.[21] On the individual differences and variations which arise in them depend the differences in activity, dilatation and stasis which were obtained.

In all cases in which duodenal ileus was produced, the primary result was a definite increase in the evacuation time of the gallbladder. The late results were variable. The possible effects of duodenal ileus on the emptying of the gallbladder are manifold:

1. Chemical irritation of the duodenum will delay the emptying of the gallbladder.[22] In our experiments, the stasis of chyme in the duodenum was never so great as to make likely any chemical irritation.

2. Acute distention of the duodenum will delay the evacution of the gallbladder. The obstructed duodenums in our experiments were usually considerably dilated, but this was a chronic and not an acute effect. It may have been a factor, however, at least in the early changes noted.

3. Dilatation of the duodenum might also be conceived to flatten out the intramural portion of the common bile duct, or to compress or distort it, and thus account for the biliary stasis. No anatomic evidence to this effect could be found, and there was no increase in the size of the common bile duct to suggest that any constant anatomic obstruction to bile flow had even recently been present.

4. Hyperperistalsis, one of the two chief reactions to duodenal ileus, would tend to increase the efficiency of the emptying of the gallbladder by: (*a*) a greater downward milking action on the intramural portion of the common bile duct and (*b*) a greater aspiration effect by the greater and more frequent changes in duodenal pressure.

Antiperistalsis is followed by the same changes in duodenal pressure, and therefore aids biliary flow by the same aspiration effects. The

20. Kellogg, E. L., and Kellogg, W. A.: Chronic Duodenal Stasis, Radiology **9**:23 (July) 1927.

21. Bloomfield, A. L., and Keefer, C. S.: Clinical Physiology of the Stomach: Simultaneous Quantitative Observations on Gastric Secretory Volume, Acidity and Motility, Arch. Int. Med. **38**:145 (Aug.) 1926. Kasabach, H. H.: Effect of Ingestion of Water and of Dextrose Solution on the Emptying Time of the Normal Stomach, ibid. **48**:1237 (Dec.) 1931.

22. Crain, R. C., and Walsh, E. L.: Effect of Acute Chemical Duodenitis upon the Emptying Time of the Gall Bladder, Surg., Gynec. & Obst. **53**:753, 1931.

peristalsis may be considered as opposing biliary flow, however, by the upward milking action.

5. Hypertonus, the second of the two chief reactions to duodenal ileus, would delay the emptying of the gallbladder by exerting a greater intramuscular tension on the portion of the common bile duct traversing it. Hypertonus and hyperactivity are roughly but not accurately correlated. The increase in peristalsis may be much greater than that in tonus, so far as the effect on biliary flow is concerned. On the other hand, tonus may be so great that peristalsis is stopped. The final effect on the biliary flow would depend on the balance between these factors.

Under the fluoroscope, between duodenal dilatation and hyperactivity, one may obtain a rough estimate of the tonus of the duodenal musculature. However, changes in tonus may well occur independently of these and may not be detectable by this method of observation.

This fact may be sufficient to account for the variability of our late results, but other factors must also be considered. Tonus changes in the sphincter of Oddi may occur independently of tonus changes in the duodenum or of peristalsis and may compensate for them. Perhaps longer contact of food in the duodenum produces more "cholecystokinin" and gives better contraction of the gallbladder. The musculature of the gallbladder, the biliary secretory pressure and the changes in the intra-abdominal pressure may determine their own compensation for whatever obstruction duodenal ileus imposes on the biliary flow.

The inconstancy of our late results is in keeping with the clinical variability of involvement of the gallbladder in duodenal ileus. In some cases biliary stasis results; in others, the duodenal changes or compensating factors are such that biliary stasis does not follow. The delay in the emptying of the gallbladder which was noted with the onset of distemper may have a clinical application. It indicates that the finding of good visualization with poor emptying of the gallbladder may be present even in extracholecystic disease.

CONCLUSIONS

1. The initial result of experimentally produced duodenal ileus in the dog is a definite increase in the evacuation time of the gallbladder.

2. When the operative procedure did not result in duodenal ileus, or if ileus later subsided, the emptying of the gallbladder was as rapid as in the preoperative controls.

3. The late results when ileus persisted were inconstant. In some dogs the delay in the evacuation of the gallbladder persisted; in others, the emptying time of the gallbladder returned to normal. This is in keeping with the clinical variability of biliary involvement in duodenal ileus. Possible explanations for these variations are discussed.

4. The symptoms of duodenal ileus produced in the dog were much less marked than those produced in man by an equivalent degree of anatomic obstruction.

5. The ability of the proximal part of the intestine to compensate for the physiologic obstruction induced by reversal of an intestinal segment in the dog is great. It is probable that this compensation occurs by an increased motor drive of the proximal intestine and perhaps also by increased antiperistalsis in the reversed segment, rather than by any actual reversal of physiologic polarity.

6. The delay in evacuation of the gallbladder noted with the onset of distemper is suggested as of clinical importance in that it is possible to obtain this finding in extracholecystic disease.

PERITONITIS

EFFECTS OF THE ADMINISTRATION OF SALT SOLUTION ON THE AMOUNT OF FLUID THAT ACCUMULATES IN THE PERITONEAL CAVITY

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There has been a growing tendency in recent years to inject large quantities of fluids intravenously in the treatment of shock, peritonitis and intestinal obstruction. MacFee and Baldridge,[1] 1930, reported their results in the treatment of such conditions by the intravenous injection of large amounts of physiologic solution of sodium chloride. They stated that: "The solution has been given intravenously in amounts ranging from 2000 cubic centimeters to 8000 cubic centimeters at a single injection. The usual amount required has been about 4500 cubic centimeters." Autopsy was performed recently on a patient who died of peritonitis and who had been given large amounts of salt solution intravenously. The peritonitis was generalized, and there was an unusually large amount of fluid in the peritoneal cavity. Following this autopsy it was feared that the extent of the peritonitis had been increased by the fluid that had been introduced and that the patient's chances of recovery had been lessened.

The present experiments were performed in order to determine the amount of free fluid that was present in the peritoneal cavities of dogs with peritonitis, to some of which no fluid had been given and in others of which fluid had been introduced intravenously or subcutaneously.

METHODS

All of the experiments were performed on dogs. Two types of slightly different experimental procedures were carried out. In the first group of experiments, the meso-appendix was ligated and divided, and an opening was made that extended half across the appendix at the junction of the proximal and middle thirds. These animals were studied until they died or until it was seen that they were going to recover. If an animal lived for less than twenty-one hours following the operation or if it recovered, the experiment was discarded. In the second group of experiments, an opening was made half across the appendix at the junction of the proximal and middle thirds. The meso-appendix was not divided. Bleeding was controlled by suture ligatures. These animals were killed, and autopsy was performed approximately forty-eight hours following the operation. The experiments in

From the Department of Surgery, Vanderbilt University.

1. MacFee, W. F., and Baldridge, R. R.: Postoperative Shock and Shocklike Conditions: Treatment by Infusion in Large Volume, Ann. Surg. **91**:329, 1930.

which the animals survived less than forty-five hours were discarded. Ether was used as the anesthetic for the operations.

The rest of the procedures were common to both types of experiments. In order to keep the animals quiet while fluids were being administered, they were given each day 0.033 Gm. of morphine sulphate prior to the beginning of the injection. In order to maintain conditions as uniform as possible and also because morphine is used in treating peritonitis, the animals not receiving fluids were also given morphine each day. No fluids or food were given by mouth following the operation. As has been stated, physiologic solution of sodium chloride was introduced intravenously in some experiments and subcutaneously in some; the remaining animals received no fluids.

The femoral vein was chosen for the intravenous injections. The subcutaneous injections were made into the tissues of the thigh and flank. The fluid was introduced each day following the operation; it was at body temperature and was given at a constant rate for a period of four hours. Ten cubic centimeters of fluid to a kilogram of body weight was given every hour for four hours.

Following the death of the animal, the body weight was determined. A midline abdominal incision was then made, and a sample of the free fluid was obtained for subsequent study. All of the free fluid was then sponged away from the peritoneal cavity. The animal was weighed again, and the difference in the weight before and after the removal of fluid was determined in grams. This figure gave approximately the number of cubic centimeters of free fluid in the peritoneal cavity.

The nitrogen content of the fluid that was obtained from the peritoneal cavity was determined by the Gunning[2] modification of the Kjeldahl method. In the tables the nitrogen is expressed as protein.

RESULTS

The percentage of deaths that occurred in less than twenty-four hours following the operation was greater in the group in which the meso-appendix was ligated and divided.

Experiments in Which the Meso-Appendix Was Severed and an Opening Made Into the Appendix.—After a few experiments had been performed, it was realized that conclusions as to the relative merits of giving no fluids and of administering salt solution subcutaneously or intravenously in preserving or lengthening the life of animals with peritonitis could not be drawn from the experimental procedures used in this work. It would require a tremendous number of experiments to determine this point. Many animals died within less than twenty hours following the operation, and since fluids were not injected until the day following operation, these experiments are not included in the tables. One or more animals in each of the three groups, namely, those receiving no fluid, those receiving salt solution intravenously and those receiving it subcutaneously, appeared to be in good condition the fourth day following the operation and were given food and water

2. Gunning, J. W.: Ueber eine Modification der Kjeldahl-Methode, Ztschr. f. anal. Chem. **28**:188, 1889.

by mouth. They recovered from the operation and are not included among those reported here.

The largest amount of free peritoneal fluid per kilogram of body weight was found in the experiments in which the salt solution was injected intravenously. The average number of cubic centimeters was 40.9. The average number of cubic centimeters per kilogram of body weight in the experiments in which no solutions were injected was 18.5, and it was 14.7 cc. in those in which the salt solution was given

TABLE 1.—*Showing the Effects of No Fluids, of Fluids Introduced Intravenously and of Fluids Injected Subcutaneously in Dogs in Which the Meso-Appendix Had Been Divided and an Opening Had Been Made into the Appendix*

Method of Administration of Fluid	Experiment	Weight of Dog, Kg.	Time Lived After Operation	Interval Between Last Fluid and Death	Total Fluid Introduced, Cc.	Fluid in Peritoneal Cavity, Cc.	Fluid in Peritoneal Cavity, Cc. per Kg. Body Weight	Protein, Gm. per 100 Cc.
No fluid	1	14.8	39 hrs.	.....	0	325	21.9	5.58
	2	13.5	24 hrs.	.....	0	180	13.3	4.53
	3	13.0	45 hrs. 30 min.	.....	0	290	22.3	6.49
	4	10.7	48 hrs.	.....	0	65	6.1	4.26
	5	15.6	37 hrs.	.....	0	480	30.7	6.30
	6	16.0	21 hrs.	.....	0	270	16.9	....
Average							18.5	5.43
Fluid intravenously	1	20.5	46 hrs. 30 min.	1 hr. 30 min.	1,640	1,400	68.3	4.59
	2	16.4	46 hrs. 30 min.	1 hr. 30 min.	1,312	500	30.5	3.24
	3	15.4	84 hrs.	12 hrs.	1,848	1,160	75.3	4.75
	4	11.4	28 hrs. 50 min.	3 hrs.	456	300	26.3	5.04
	5	5.8	30 hrs. 50 min.	10 hrs.	230	140	24.1	6.50
	6	9.6	84 hrs.	12 hrs.	1,153	200	20.8	6.81
Average							40.9	5.16
Fluid subcutaneously	1	12.1	45 hrs.	20 hrs.	484	180	14.9	5.83
	2	16.1	30 hrs. 45 min.	7 hrs.	644	160	9.9	5.81
	3	13.5	31 hrs.	8 hrs. 30 min.	540	305	22.6	5.56
	4	8.1	22 hrs.	1 hr.	324	80	9.9	....
	5	9.3	60 hrs.	12 hrs.	744	135	14.5	5.59
	6	11.2	24 hrs.	0 hrs.	400	170	15.2	....
	7	7.9	49 hrs.	1 hr.	633	125	15.8	4.65
Average							14.7	5.49

subcutaneously. It is surprising that there was a slightly larger amount of free peritoneal fluid in the experiments in which no fluid was injected than in those in which it was given subcutaneously, and this probably would not have been the case if the number of experiments had been greater. This finding was reversed in the second type of experiments to be reported later in this paper. There was little difference in the average protein content of the peritoneal fluid in the three groups of experiments. The lowest values were found in the experiments in which the fluid was given intravenously.

The results of these experiments are given in table 1.

Experiments in Which an Opening Was Made Into the Appendix.—The experiments were discarded in which at the expiration of the forty-eight hour period no free fluid was found in the peritoneal cavity. Such a finding was the exception rather than the rule. The largest amount of free fluid was found in the experiments in which salt solution was injected intravenously. The average number of cubic centimeters per kilogram of body weight in these experiments was 34. The corresponding figure in the experiments in which fluid was given

TABLE 2.—*Showing the Effects of No Fluids, of Fluids Introduced Intravenously and of Fluids Injected Subcutaneously in Dogs in Which an Opening Had Been Made into the Appendix. Dogs Killed Approximately Forty-Eight Hours After Operation*

Method of Administration of Fluid	Experiment	Weight of Dog, Kg.	Length of Experiment, Hours	Total Fluid Introduced, Cc.	Fluid in Peritoneal Cavity, Cc.	Fluid in Peritoneal Cavity, Cc. per Kg. Body Weight	Protein, Gm. per 100 Cc.
No fluid	1	14.8	46	0	25	1.7	7.66
	2	18.5	52	0	275	14.9	7.41
	3	19.0	51	0	135	7.1	5.75
	4	11.5	51	0	140	12.2	6.32
	5	10.9	50	0	55	5.0	5.91
Average						8.2	6.61
Fluid intravenously	1	10.1	49	808	670	66.3	5.55
	2	11.0	49	880	260	23.6	2.73
	3	12.8	45	1,024	565	44.1	4.63
	4	8.6	45	680	165	19.2	5.06
	5	8.9	51	712	150	16.9	5.28
Average						34.0	4.65
Fluid subcutaneously	1	9.5	46	756	115	12.1	5.63
	2	9.3	46	746	170	18.3	5.35
	3	8.5	45	673	170	20.0	5.00
	4	10.5	49	842	35	3.3	4.73
	5	7.0	45	556	75	10.7	5.23
	6	10.3	50	821	130	12.6	4.70
	7	11.5	45	920	100	8.7	5.14
Average						12.2	5.11

subcutaneously was 12.2 cc., and in those in which no fluid was given, it was 8.2 cc. The protein content of the free peritoneal fluid was highest in the experiments in which no fluid was given, and it was lowest in the experiments in which salt solution was injected intravenously. The results of these experiments are given in table 2.

COMMENT

From these experiments, conclusions cannot be drawn as to whether the introduction of salt solution intravenously increases or decreases the chances of recovery of an animal with peritonitis. The fact that the animals which received salt solution intravenously had on the average

approximately three times as much free fluid as those getting no fluids and those receiving salt solution subcutaneously suggests that the introduction of large amounts of fluids intravenously in the presence of peritonitis may be harmful. If the circulation is in sufficiently good condition to absorb fluids, it is probably better to administer them by some route other than the intravenous one. In the present experiments the introduction of salt solution subcutaneously was associated with approximately the same amount of free peritoneal fluid as the giving of no fluids. The effects of the introduction of fluids by rectal tube were not studied, because usually dogs expel fluid administered by this method.

Beard and Blalock[3] found that the introduction of physiologic solution of sodium chloride intravenously while the intestines were being traumatized was associated with the escape of an unusually large amount of fluid into the peritoneal cavity. The present experiments show that bacterial and mechanical injuries act similarly in this respect.

SUMMARY

The effects on the amount of free peritoneal fluid and on the protein content of this fluid of not giving fluids, of injecting salt solution intravenously and of introducing salt solution subcutaneously have been determined in dogs with peritonitis. No food or water was given by mouth. In the first group of experiments, peritonitis was produced by ligating and dividing the meso-appendix with its vessels and by making an opening into the appendix. These animals were observed until death or recovery took place. In the second group an opening was made into the appendix, and bleeding vessels were ligated. The experiment was terminated approximately forty-eight hours later.

The animals which received salt solution intravenously had an average of approximately three times as much free peritoneal fluid as those getting no fluids and those receiving salt solution subcutaneously. The content in protein of the free peritoneal fluid was lowest in the animals in which salt solution was given intravenously.

3. Beard, J. W., and Blalock, Alfred: Intravenous Injections: A Study of the Composition of the Blood During Continuous Trauma to the Intestines When No Fluid is Injected and When Fluid is Injected Continuously, J. Clin. Investigation **11**:249, 1932; The Effects on the Composition of the Blood of the Subcutaneous Injection of Normal Salt Solution into Normal Dogs and into Dogs Subjected to Intestinal Trauma, Graded Hemorrhages and Histamine Injection, ibid. **11**:311, 1932.

EXPERIMENTAL PERITONITIS

THE RÔLE OF THE WELCH BACILLUS

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Probably the most serious types of peritonitis are those in which not only virulent micro-organisms are present in the peritoneal cavity, but, in addition, culture material for the micro-organisms to develop on. This set of conditions is seen in various types of perforative peritonitis or in lesions accompanied by the death of tissue, such as postoperative peritonitis or peritonitis with strangulated hernia. Since it is difficult to produce peritonitis experimentally by the introduction of bacteria alone into the normal peritoneum, it seemed logical to assume that experimental peritonitis could be produced by the introduction of bacteria growing on a culture medium. By such a method it would be possible, theoretically, to study peritonitis produced by a single type of micro-organism or by selected groups of micro-organisms. Accordingly, pure cultures of Bacillus coli, staphylococcus, streptococcus and pneumococcus on agar or blood agar slants were introduced into the peritoneal cavity of dogs and rabbits. Many of these animals became extremely sick, and most of them died or were killed within twenty-four hours. At autopsy, in addition to the original micro-organism introduced into the peritoneal cavity, B. welchii was found to be present, and often predominated in numbers. In some instances, streptococci or staphylococci were found to be present when the original organism introduced was B. coli. The peritoneum was reddened and contained a small amount of serosanguineous fluid with a little fibrin. Blood cultures were not taken in all experiments, but when cultured, they were negative. Control experiments in which plain agar was introduced into the peritoneum gave negative results. A summary of these experiments follows.

EXPERIMENTATION

Medium-sized dogs were selected, and under careful aseptic technic agar slants with a twenty-four hour growth of colon bacillus, streptococcus, staphylococcus or pneumococcus were placed within the peritoneal cavity through a small, lower rectus incision. Seventeen

From the laboratories of Rush Medical College.

of the 23 dogs died within from twelve to thirty-six hours of an extensive hemorrhagic peritonitis. The results of this series are given in table 1.

Postmortem examination was made immediately or a few hours after death. Several of the dogs were markedly distended, and gas escaped from the peritoneal cavity when the abdomen was opened. Smears and aerobic and anaerobic cultures were made from the peritoneal fluid. A gram-positive anaerobic bacillus, which was identified as the Welch bacillus, was cultured along with the bacterium that had been growing on the agar slant. Other bacteria were cultured from 6 of the 17 dogs with peritonitis.

TABLE 1.—*Agar Slant and Bacteria Given Intraperitoneally* *

Bacteria	Dogs	Deaths	B. Welchii	Other Bacteria
Colon bacillus	12	9	8	Streptococcus, 1
Streptococcus	5	3	3	Staphylococcus, 1
Staphylococcus	3	2	2	Streptococcus, 2
Pneumococcus	3	3	3	Streptococcus, 2
	Rabbits			
Colon bacillus	4	2	2	None
Streptococcus	2	1	1	None
Staphylococcus	2	1	1	None

Sterile Agar Slant Intraperitoneally

Dogs	Deaths	Postmortem	Cultures
6	0	Negative	Sterile

Physiologic Solution of Sodium Chloride and Colon Bacillus Intraperitoneally

Dogs	Deaths	Postmortem	Cultures
2	0	Negative	Sterile

* The same strain of B. coli was used on an agar slant for introduction into the peritoneum of 2 dogs; death occurred within twenty-four hours of peritonitis from which the Welch bacillus was cultured.

Four dogs were killed after sixteen hours to rule out the possibility of postmorten invasion of the Welch bacillus.

In 6 dogs sterile agar slants were placed intraperitoneally. Postmortem examination of these dogs showed the agar encapsulated, and the cultures were sterile.

The same strain of B. coli that produced a hemorrhagic peritonitis with the invasion of the Welch bacillus was injected intraperitoneally in a physiologic solution of sodium chloride into 2 dogs. Both dogs survived but were killed forty-eight hours later; the peritoneal fluid was sterile.

To rule out the possibility that these experiments were peculiar to dogs, agar slants with B. coli, streptococcus or staphylococcus were placed in the peritoneal cavity of eight rabbits. Four of the rabbits died of peritonitis; Welch bacillus and the bacterium from the agar slant were cultured.

The suspicion arose that the secondary invaders into the peritoneum might come from the nearby intestine by continuity due to damage or inflammation of the wall of the bowel. Accordingly, under aseptic technic a piece of muscle was removed from the abdominal wall or from the hind leg of a dog. In the defect was placed an agar slant with a twenty-four hour culture of the colon bacillus, streptococcus, staphylococcus or pneumococcus. Agar slants with bacteria were also placed between muscle bundles without removal of any of the muscle. Thirteen dogs were used in this group; two deaths occurred. Cultures from the pus of all the wounds showed the Welch bacillus along with the bacterium from the agar slants. Blood cultures were sterile. Gas was palpated in all the wounds. The muscle removed was passed through a Rosenau grinder, and the cultures were found to be sterile.

TABLE 2.—*Agar Slant with Colon Bacillus in the Abdominal Wall* *

Dogs	Deaths	B. Welchii
3	0	2

Sterile Agar Slant in Abdominal Wall

Dogs	Deaths	Cultures
3	0	Sterile

Agar Slant with Bacteria in Muscle of Leg

Bacteria	Dogs	Deaths	B. Welchii
Colon bacillus	4	1	4
Staphylococcus	2	1	2
Streptococcus	2	0	2
Pneumococcus	2	0	2

* A sterile agar slant was placed in the other hind leg of all the dogs; it always remained sterile.

At the time when the agar slant with a bacterial growth was placed intramuscularly in the hind leg of a dog, a sterile agar slant was placed in a corresponding position in the opposite leg. Also, in 3 dogs sterile agar slants were placed between the muscles of the abdominal wall. The dogs were killed forty-eight hours later, and the cultures were sterile.

These experiments are summarized in table 2.

In order to determine whether local infection was the essential factor in creating conditions favoring the appearance of secondary invading micro-organisms, several types of nonbacterial inflammatory reactions were set up in the peritoneal cavity.

A 50 per cent solution of dextrose, a 5 per cent solution of turpentine emulsion and a 0.3 per cent solution of hydrochloric acid were injected intraperitoneally, and the dogs were killed at the end of twenty-four hours. Postmortem examination showed a slightly inflamed peritoneum with a transudate, but aerobic and anaerobic cultures were

sterile. The possibility of producing peritonitis by chemical irritation of the peritoneum in the presence of mediums for bacterial growth was tried. Sterile agar slants were placed in the abdominal cavity under careful surgical technic along with dextrose or the turpentine solution. The dogs were killed twenty-four hours later, and the cultures were sterile.

Steinberg and Goldblatt [1] reported that they were able to produce fatal peritonitis by the intraperitoneal injection of 40 cc. of a 2.5 per cent solution of tragacanth with B. coli. We repeated their work using the colon bacillus, streptococcus and staphylococcus in 9 dogs; 6 deaths occurred from hemorrhagic peritonitis. Cultures of the peritoneal fluid contained the Welch bacillus along with the bacterium injected. Sterile

TABLE 3.—*Peritoneal Irritation*

1. Dextrose: 50 cc. of a 50 per cent solution: 2 dogs
2. Dextrose: 50 cc. of a 50 per cent solution with 10 cc. of 2.5 per cent solution of iodine: 2 dogs
3. Dextrose and a sterile agar slant: 1 dog
4. Turpentine and a sterile agar slant: 2 dogs
5. 0.3 solution of hydrochloric acid at two hour intervals: 3 dogs

All dogs survived. Postmortem examinations were negative except for a transudate and adhesions.

40 Cc. of a 2.5 Per Cent Solution of Tragacanth with Bacteria Administered Intraperitoneally

Bacteria	Dogs	Deaths	B. Welchii
Colon bacillus	4	4	4
Streptococcus	2	1	1
Staphylococcus	3	1	1

40 Cc. of a 2.5 Per Cent Solution of Tragacanth Administered Intraperitoneally

Dogs	Deaths	Postmortem	Cultures
3	0	Negative	Sterile

tragacanth injected into 3 dogs had no effect, and the cultures of the peritoneal fluid were sterile.

The results of these experiments, summarized in table 3, show that chemical inflammation of the peritoneum is not followed by the appearance of bacteria in the inflamed area.

Therefore, it can be assumed from these experiments that the secondary invading micro-organisms were not introduced into the tissue from without and were not part of an accompanying blood stream infection; that they did not come directly by extension from the intestinal tract into the peritoneal cavity, and that they therefore must have been present in the tissue at the beginning of the experiment. It seems fair to assume, then, that B. welchii and occasionally other micro-organisms must be seeded into the different tissues of the body in laboratory animals through the blood stream without causing local

1. Steinberg, B., and Goldblatt, H.: Studies on Peritonitis, Arch. Int. Med. **39**:446 (March) 1927.

manifestations until the local resistance of the tissue is lowered by infection, when B. welchii rapidly overgrows the original infection, often with a fatal result.

How do these experimental facts square themselves with clinical and experimental knowledge of this subject, especially as related to anaerobic bacteria?

The Presence of B. Welchii in Human Feces.—As the intestinal tract is the most usual site for B. welchii, it is worth noting that Dudgeon [2] found that in 200 specimens of human feces, B. welchii was present in 35 per cent. Dudgeon also stated that in acute intestinal obstruction and in vomiting accompanying advanced peritonitis, the vomitus contained B. welchii in most instances.

The Presence of Micro-Organisms in the Organs and Blood of Freshly Killed Animals.—In 1909, Conradi [3] examined 162 organs from freshly slaughtered cattle and pigs; 72 of them contained micro-organisms. Bacteria were found in 42 of 63 livers, 18 of 59 muscles, 6 of 19 kidneys, and 4 of 5 lungs. The micro-organisms were both aerobic and anaerobic. The latter were present in 30 per cent of the tissues examined.

Reith [4] found that 77 per cent of 216 slaughtered hogs had bacteria in the muscle, and of these, 37 per cent were anaerobes.

Experimental Studies on the Ability of Micro-Organisms to Pass out of the Intestinal Tract into the Lymph or Blood Stream.—Arnold's [5] many contributions to the subject show that when the upper half of the intestinal tract in dogs, which is normally slightly acid, is made alkaline, the intestinal flora of the large bowel is found. Working with injection of B. prodigiosus into the duodenum, with or without accompanying egg white, he found that bacteria went into the blood stream quickly and in great numbers in the presence of egg white in the duodenum.

In the examination of mesenteric lymph glands in dogs, 50 per cent were found to contain a varied bacterial flora, which was greatly increased in obstruction of the bowel (Dr. David).

Presence of Micro-Organisms in the Urine in Patients with Focal Infections in the Mouth.—G. F. Dick and G. R. Dick [6] found that 60 per cent of patients examined by them with evident foci of infection had considerable numbers of bacteria in the urine. In a number of instances one or more of the kinds of bacteria found in the urine were isolated from the foci of infection in the mouth.

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2. Dudgeon, L. S.: J. Hyg. **25**:119, 1926.
3. Conradi, H.: München. med. Wchnschr. **56**:1318, 1909.
4. Reith, A. F.: J. Bact. **12**:367, 1926.
5. Arnold: J. Hyg. **29**:82, 1931; Proc. Soc. Exper. Biol. & Med. **28**:358, 1931.
6. Dick, G. F., and Dick, G. R.: The Bacteriology of the Urine in Focal Infections, Arch. Int. Med. **19**:493 (March) 1917.

Liver Autolysis Experiments.—It has been shown that small pieces of dog's liver placed in the peritoneal cavity of dogs caused death of the animals. Ellis and Dragstedt [7] showed that a gram-positive anaerobe was present in the peritoneum of these dogs. If, however, liver from pups removed by cesarean section were placed in the peritoneum no ill effects were noted, and the culture of the liver was sterile. Andrews [8] repeated the experiments, and concluded that B. welchii probably passed through the intestinal wall and infected the liver. Later, Rewbridge [9] and Andrews, Rewbridge and Hrdrina [10] allowed bile to flow into the peritoneal cavity or injected sterile bile salts into the peritoneum, and concluded that death was not due to toxemia from bile salts but to a B. welchii infection. They also took 100 Gm. of ground autoclaved dog's muscle and implanted it into the peritoneal cavity of dogs. They found at autopsy that all had B. welchii infection. They concluded that B. welchii was normally present in dog's muscle, and that injection of sterile bile salts into the muscle would activate the B. welchii so that a fatal infection ensued.

Clinical Investigations on the Presence of B. Welchii in the Soft Parts and Peritoneum.—1. It has been frequently noted, especially during the war, that fatal gas bacillus infection was often due to B. welchii growing in symbiosis with the streptococcus.

2. The presence of B. welchii in appendicitis and peritonitis has been noted. Heyde [11] studied the anaerobic flora in 102 acutely inflamed appendixes, most of which were perforated. In 100 he found anaerobes, of which 15 per cent were B. welchii, 20 per cent anaerobic streptococci and 45 per cent small gram-negative anaerobes.

Weinberg, Prevet, Davisme and Ranard [12] stated that in the study of 160 acutely inflamed appendixes the anaerobes predominated in the gangrenous cases. Thirty-three per cent were B. welchii, 39 per cent an anaerobic gram-negative bacillus and 19 per cent anaerobic cocci.

Meleney, Harvey and Zern [13] found that in cases of perforated appendixes with diffuse peritonitis B. welchii was present in 27 per cent, streptococci in 55 per cent, and B. coli in 100 per cent. In acute non-perforated local peritonitis, B. welchii was present in 13 per cent, in acute diffuse peritonitis in 31 per cent, and in appendical abscesses in 44

7. Ellis, J. C., and Dragstedt, L. R.: Liver Autolysis in Vivo, Arch. Surg. **20**:8 (Jan.) 1930.

8. Andrews, Edmund: Proc. Soc. Exper. Biol. & Med. **27**:987, 1930.

9. Rewbridge: Surg., Gynec. & Obst. **52**:205, 1931.

10. Andrews, Rewbridge and Hrdrina: Surg., Gynec. & Obst. **53**:176, 1931.

11. Heyde: Beitr. z. klin. Chir. **76**:1, 1911.

12. Weinberg, Prevet, Davisme and Ranard: Ann. Inst. Pasteur **42**:1167, 1928.

13. Meleney, F. L.; Harvey, H. D., and Zern, H. Z.: Peritonitis, Arch. Surg. **22**:1 (Jan.) 1931.

per cent. In all of these conditions, B. coli and streptococci were present in varying percentages.

Eichhoff and Pfannensteil [14] showed that B. coli, streptococcus and Frankel's bacillus were the predominating bacteria in appendicitis. In acute appendicitis, Frankel's bacillus was present in 33 per cent.

Bacteriology of Perforated Duodenal Ulcer.—Lohr [15] found that in early perforated duodenal ulcer the peritoneal cavity contained no pathogenic micro-organisms. In 22 patients that he examined B. coli were not found up to twelve hours after perforation. Later, however, the same type of micro-organisms that are found in the colon appear in the peritoneal exudate. Anaerobes, however, are rare.

COMMENT

The experiments reported in this paper lead one to believe that in certain laboratory animals local infections from known bacteria are early changed in character by the appearance in the lesion of other bacteria, generally B. welchii, which soon predominate over the original infection. The source of the secondary invading bacteria seems to be the local tissue, and it is probable that these bacteria were there in an inactive and nonpathogenic form until brought to life by the local infection, which created favorable conditions for their growth.

In this connection it should be noted that the culture of dog's muscle on artificial mediums failed to develop bacteria, whereas in the living animal muscle, micro-organisms growing on culture mediums created such favorable conditions for growth that the attenuated bacteria already in the muscle became activated and overgrew the bacteria used in the experiment. To what extent B. welchii gains access to the blood stream from time to time in the dog's intestinal tract and becomes seeded throughout the body tissues is not known. But some such distribution of B. welchii in dog and rabbit tissue seems probable from these experiments. Finally, it seems probable that bacteria are present in an avirulent state in animal tissue, and that they may rapidly assume a virulent character if a continuing infection develops from bacteria introduced into the tissues from without.

To what extent these findings and conclusions can be applied to infections in human tissues is, of course, a conjecture. Certain facts, however, make it an interesting speculation. There are many examples of the insidious migration and lodging of bacteria in the tissues of the human body, such as arthritis, osteomyelitis and cholecystitis. It is also known that bacteria may be dormant in tissues for long periods without producing clinical symptoms until, as a result of trauma or favorable

14. Eichhoff and Pfannensteil: Beitr. z. klin. Chir. **151**:171, 1931.

15. Lohr: Zentralbl. f. Chir. **53**:1618, 1926; Ztschr. f. Chir. **214**:103, 1929.

local conditions, they become active, as in the case of ankylosed joints following suppurative arthritis. Evidence also exists that some bacteria seem to flourish best when growing in symbiosis with other bacteria, as for instance in peritonitis or gas bacillus infection. The great variety of bacteria found in peritonitis, cholecystitis and cellulitis (as ischiorectal abscess) offers the opportunity to consider whether some of the bacteria found in these lesions were lying dormant in the tissues where they had previously lodged and were activated by the introduction of an outside or new infection in these tissues. The presence of streptococci in so many lesions may possibly be accounted for on a similar basis. It is difficult to prove such a contention; we have no direct proof, and only offer the possibility as an analogy to the experimental data on animals.

CONCLUSIONS

The introduction of bacteria on culture mediums into the tissues of certain animals results in severe infection which is due not only to the bacteria introduced but to secondary invaders, chiefly B. welchii. Experimental evidence is submitted which indicates that the secondary invading bacteria were already present in the tissue in an avirulent state but were activated to growth by the infection introduced.

The practical significance of these observations concern: 1. The importance of the death of tissue in surgical procedures acting as a favorable culture medium for the development of bacteria already present in the tissue or introduced at the time of the operation.

2. The suggested mechanism of the development of some secondary infections.

3. The increase of the virulence of B. welchii when growing in symbiosis in living tissue with pus-producing bacteria.

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DIVERTICULITIS OF THE COLON IN WOMEN

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The recognition of the clinical importance of diverticulitis of the colon is largely a development of the past two decades. Even though Virchow,[1] in 1853, described "isolated, circumscribed, adhesive peritonitis" of the colon, it was not until 1899 that Graser,[2] as the result of postmortem studies, directed attention to the association of these localized inflammatory processes with diverticula of the large bowel. In 1907, Mayo, Wilson and Giffin [3] reported five cases of diverticulitis that had been encountered at operation and emphasized for the first time the symptomatology of the disease. It was not until 1914 that the condition was recognized by roentgenologists.[4] Since that time the ever increasing number of reports of this condition in medical, surgical and roentgenologic literature has brought convincing evidence of its enlargeing clinical importance.

Diverticulitis of the colon may be defined as an inflammation of one or more diverticula, or accessory pouches, of the large bowel. The term diverticulosis is usually employed to describe the presence of asymptomatic diverticula. True diverticula of the colon are exceedingly rare and are of little clinical importance; such diverticula are congenital and represent a developmental disturbance in which the walls of the pouches are composed of mucosa, muscularis and serosa. The most common form of diverticulum is the false or acquired diverticulum, which apparently has its origin in a structural weakness of the muscularis that permits the mucosa to herniate through the divided muscular layer and form a pouch, the wall of which is made up of mucosa and serosa. Such acquired diverticula have been aptly called mucosal hernias.

The great majority of diverticula occur in the sigmoid colon. In an operative series of forty-two cases of diverticulitis of the colon reported

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From the Surgical Division of the Dayton Clinic.

1. Virchow, R.: Historisches, kritisches und positives zur Lehre der Unterleibsaffektionen, Virchows Arch. f. path. Anat. u. Physiol. **5**:281, 1853.

2. Graser, E.: Ueber multiple Darmdivertikel in der Flexura sigmoidea, Verhandl. d. deutsch. path. Gesellsch. **28**:254, 1899; Das falsche Darmdivertikel, Arch. f. klin. Chir. **59**:638, 1899.

3. Mayo, W. J.; Wilson, L. B., and Giffin, H. Z.: Acquired Diverticulitis of the Large Intestine, Surg., Gynec. & Obst. **5**:8, 1907.

4. Abbe, R.: Case of Sigmoid Diverticulitis Simulating Malignancy, M. Rec. **86**:190, 1914.

by W. J. Mayo,[5] sigmoid diverticula were found in thirty-six cases. Judd and Pollock [6] found diverticulitis of the sigmoid colon in 75 per cent of one hundred and eighteen cases, while Masson [7] found the colonic diverticula to be restricted to the sigmoid colon in 81 per cent of one hundred and sixteen cases. In about 20 per cent of cases, diverticula may be found in the cecum, ascending colon, transverse colon, descending colon or in the rectum.

Diverticula may vary in number from one to several hundred. In rare instances the entire colon may be studded with saccules varying in size from that of a pinhead to that of a walnut. There is no agreement as to any constant location of diverticula in their relation to the mesentery. Some writers have stated that diverticula are most commonly observed at the antimesenteric border, particularly at the point of penetration by blood vessels. As a matter of fact, diverticula have been found to occur at any point along the circumference of the wall of the large bowel. Kieth [8] expressed the opinion that diverticula result from increased intracolonic pressure, with the protrusion of sacculations of mucous membrane through weak areas in the musculature. In all probability the production of acquired diverticula is dependent on several factors. The disease occurs most commonly in obese persons past 40 years of age, who are physically inactive and who have experienced chronic constipation for many months or years. Ordinarily, chronic constipation affects the sigmoid colon to a greater degree than it does other parts of the large bowel, because the fecal current is slowest in the sigmoid flexure. It seems probable, therefore, that a combination of some or all of these circumstances is responsible for the development of diverticula. Diverticulitis is much more common in men than in women, in the ratio of approximately 2:1.

That diverticulosis and diverticulitis are much more common than is generally believed is illustrated by the frequency with which diverticula are discovered accidentally as the result of routine roentgen studies. At Washington University, Larimore [9] found definite evidence of diverticulosis in fifty five (1.65 per cent) of 4,408 patients who were subjected to roentgen studies of the intestinal tract. Spriggs and Marxer [10] found

5. Mayo, W. J.: Diverticulitis of the Large Intestine, J. A. M. A. **69**:781 (Sept. 8) 1917.

6. Judd, E. S., and Pollock, L. W.: Diverticulitis of the Colon, Ann. Surg. **80**:425, 1924.

7. Masson, J. C.: Diverticulitis of the Large Bowel, Canad. M. A. J. **11**:106, 1921.

8. Kieth, A.: Diverticula of the Alimentary Tract of Congenital or of Obscure Origin, Brit. M. J. **1**:376, 1910.

9. Larimore, J. W.: Diverticulitis of the Large Intestine, J. Missouri M. A. **22**:129, 1925.

10. Spriggs, E. I., and Marxer, O. A.: Intestinal Diverticula, Brit. M. J. **1**: 130, 1926.

one hundred cases of diverticulosis in 1,000 roentgenologic examinations of the large bowel.

DIAGNOSIS AND TREATMENT

Diagnosis.—Diverticulitis may manifest itself as an acute or chronic disease. When the disease exists in the chronic form, acute exacerbations are common. Ordinarily the patient will complain of vague, intermittent pain, occasionally cramplike in severity, in the lower left abdominal quadrant. Occasionally the pain is most pronounced in the lower mid-abdominal region. Many patients will complain of bladder symptoms, notably frequency and tenesmus. If the abdominal wall is thin, it is usually possible to palpate a tender, ropelike, hard, nodular sigmoid colon. The sacculations usually become filled with fecal material which becomes inspissated and often impregnated with calcium salts, forming rocklike fecaliths. These scybalous masses may cause pressure atrophy and erosion of the intestinal wall, with the development of perforation, abscess or localized or generalized peritonitis. In some instances fistulas may develop into the urinary bladder or through the skin. The development of large inflammatory masses with or without perforation may produce angulation and obstruction. In rare instances adenocarcinoma may develop in the diverticulum, probably as a result of prolonged irritation.

Acute diverticulitis usually produces the symptomatology of left-sided appendicitis. The severe sudden abdominal pain is usually generalized at the abrupt onset of illness, but becomes localized in the region of the suppurative diverticulitis. These symptoms are accompanied by localized tenderness, muscle spasm, fever and leukocytosis; nausea and vomiting may occur. While the suppurative process remains restricted to the diverticulum, the fever and leukocytosis are usually of the mild degree seen in acute appendicitis. Extensive ulceration of the mucosa of the diverticulum is rare, so that it is unusual to find blood in the stool.

Unless the symptoms indicate that immediate operation is advisable, roentgen studies should be made. The roentgenographic appearance of diverticula of the large bowel is usually characteristic and diagnostic. If the saccules are filled with fecal matter, it is often impossible to outline the diverticula with the barium mixture. A ragged, spastic, "saw-tooth-like" appearance of the large bowel is highly suggestive and should invite further roentgen studies.[11] If roentgenograms are taken from twenty-four to seventy-two hours after the opaque meal is given, the residue of barium will frequently outline the pockets after the lumen of the bowel has been emptied.[12]

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11. Mackoy, F. W.: Family Diverticulosis of the Colon, Radiology **7**:498, 1926.
12. Enfield, C. D.: Diverticulosis and Diverticulitis of the Colon, Am. J. Roentgenol. **12**:242, 1924.

Treatment.—The mere presence of symptomless diverticula does not warrant surgical intervention. Surgical treatment should be confined to acute diverticulitis or to the complications of chronic diverticulitis. The most common complications are perforation, with the development of localized or generalized peritonitis, formation of abscesses, intestinal obstruction due to a large inflammatory mass, fistulas between the involved area and the bladder, intestine or skin, secondary abscess of the liver and a malignant condition. Perforation of a diverticulum during an acute attack is rare. Ordinarily, the perforation does not occur into the free peritoneal cavity, because the preexisting inflammatory reaction usually fixes the sigmoid to loops of the small intestine or to the parietal peritoneum or anterior abdominal wall. Consequently, formation of abscess is the most common complication.

The development of acute diverticulitis requires surgical treatment. If it is found that localized peritonitis or abscess has developed, the area should be drained, and the loop of bowel containing the infected area should be drawn through a split-muscle incision. I have found it unwise to excise or open this loop because of the danger of rupture of the diverticulum and the escape of fecal material. If the diverticulum should rupture following its withdrawal from the abdominal cavity, little or no harm results. Hot boric acid dressings should be applied to the exposed loop of infected bowel until the infection has become inactive. After the inflammatory process has entirely subsided, it is possible to release the loop of bowel and place it beneath the abdominal muscles, external to the peritoneum. The incision is then closed without drainage. If the diverticulitis does not subside, a Mikulicz resection usually yields the best results.

On opening the abdomen it may be difficult in certain instances to distinguish between carcinoma of the large bowel and diverticulitis. The regional lymph-nodes are usually enlarged and firm in the presence of diverticulitis, as well as of carcinoma. In such cases a rapid microscopic examination of the tissue should be carried out. If any doubt exists as to the presence of carcinoma, radical resection is indicated. The loop of bowel containing the neoplasm can usually be delivered through the abdominal incision, and a Mikulicz resection can then be performed.

REPORT OF CASES

One purpose of this undertaking is to present the pertinent clinical findings in four cases of diverticulitis of the colon, three of which presented pelvic symptoms that led to confusion with gynecologic conditions.

CASE 1.—Mrs. N. A., aged 52, an obese housewife, presented herself on July 1, 1930. With the exception of chronic constipation, she had enjoyed excellent health until four weeks before this time. The menstrual history had been entirely uneventful prior to the onset of the present illness. Four weeks previously the patient began to experience continuous pain in the lower part of the abdomen,

which became localized in the right lower quadrant. About one week following the development of the abdominal pain the patient began to have continuous vaginal bleeding, which gradually increased in intensity. She had lost 30 pounds (14 Kg.) in weight during the four weeks of illness. On examination, the temperature was found to be 100 F., with a heart rate of 92. The skin and mucous membranes showed marked pallor. The tongue was dry and parched. Examination of the head, neck and chest revealed no abnormalities. A large nodular mass was found in the center of the lower part of the abdomen, which extended 3 cm. above the umbilicus. The mass was exquisitely tender on pressure. On vaginal examination a large, tender, fixed mass was found to fill the entire pelvis.

Hematologic studies revealed 2,630,000 red blood cells per cubic millimeter and 2,300 white blood cells, with 93 per cent neutrophilic polymorphonucleosis. The estimation of hemoglobin was 45 per cent (Dare). Chemical analysis of the blood showed: creatinine, 2 mg. per hundred cubic centimeters; urea nitrogen, 17 mg.; blood sugar, 357 mg., and blood chlorides, 505 mg. The Wassermann and Kahn tests of the blood were negative. An analysis of the urine revealed a large amount of albumin and 2.1 Gm. of sugar per hundred cubic centimeters, with acetone and diacetic acid. The microscopic examination revealed a large number of separate and clumped pus cells; there were no red blood cells and no casts.

The patient was given medical treatment, which consisted of regulation of the diet, insulin, forced fluids and transfusions. During the following week, the blood sugar was restored to the normal level, and three transfusions were given. Laparotomy was done on July 8, 1930, with the preoperative diagnosis of multiple fibromyomas of the uterus. A large nodular uterus, containing multiple fibromyomas, was found, with an orange-sized abscess between the omentum and the fundus of the uterus, which extended into the left broad ligament and involved the sigmoid colon. This colon showed multiple diverticula. The abscess originated in one of the diverticula. Supravaginal hysterectomy, bilateral salpingectomy and appendectomy were done. The loop of sigmoid colon containing the infected diverticulum was brought to the outside of the abdomen through a split-muscle incision. Dressings of hot boric acid were repeatedly applied to the exposed portion of the sigmoid colon, and after two weeks the inflammatory process had entirely subsided. Three days after the sigmoid had been withdrawn from the abdomen, the diverticulum ruptured and discharged purulent fecal material for five days. After the inflammatory process had subsided, the sigmoid was detached from the skin and placed beneath the left rectus muscle, external to the peritoneum. The convalescence was uneventful, and the patient was discharged from the hospital on Aug. 17, 1930. She has since regained her normal weight and has been in excellent health.

CASE 2.—Mrs. D. K., an obese housewife, aged 54, was first seen on Nov. 3, 1928. Her health had always been excellent. She had experienced chronic constipation for many years. The menopause had occurred six years previously. Three weeks before admission to the clinic she experienced a sudden, severe pain in the lower left abdominal quadrant, which was relieved following an enema. Since that time the pain had persisted to a less marked degree in the same region. Occasionally it became generalized over the entire abdomen, and she became nauseated. The general physical examination revealed nothing of importance except in the abdomen and pelvis. A tender mass, the size of a lemon, was palpable in the lower left abdominal quadrant. There was no rigidity of the muscle. On vaginal examination the uterus was found to be small and tender on deep pressure. There was a tender, palpable mass, the size of an orange, in the region of the left broad ligament.

The temperature was 99.6 F., with a pulse rate of 95. Hematologic studies revealed a slight degree of secondary anemia, with no leukocytosis. The Wassermann and Kahn tests of the blood were negative. Urinalysis gave negative results.

On Nov. 4, 1928, laparotomy was performed, with the preoperative diagnosis of pelvic tumor. A large orange-sized, fluctuant mass was encountered at the rectosigmoid junction. About 3 inches (7.6 cm.) above this mass and connected with the sigmoid colon was a similar mass about 5 cm. in diameter. The gross appearance strongly suggested carcinoma of the sigmoid. The first stage of a Mikulicz operation was performed. The pathologist reported multiple diverticula, with no evidence of malignant transformation. Hot boric acid dressings were applied to the intestine; fourteen days later it was placed beneath the rectus muscle, external to the peritoneum, and the abdominal wound was closed.

On Nov. 12, 1928, the patient was discharged from the hospital with a small draining sinus, which closed within the next two weeks. On Feb. 27, 1930, a barium enema was given; the roentgenograms revealed a normal colonic outline, except for the presence of several small pea-sized diverticula near the splenic flexure. At present the patient is in excellent health.

Case 3.—Mrs. M. S., a housewife, aged 48, presented herself for clinical study on Jan. 31, 1931. The chief complaint was abdominal pain. During the summer of 1930, the patient had experienced severe "bearing down" pains in the abdomen and pelvis. Her previous health had been good, except for an almost constant dull headache that had persisted for years. Coincident with the development of the abdominal pain, urinary frequency and nocturia developed. The patient was unable to empty the bladder completely. These symptoms persisted until November, 1930, when they abruptly ceased. The previous good health was regained, except for constant backache.

On the day before admission to the clinic acute pain developed in the lower part of the abdomen, associated with marked urinary frequency. The pains were sharp and cramping. She had always been more or less obese and had experienced no recent loss in weight. There had been no menstrual difficulties prior to the menopause, which occurred at 44 years. She had four healthy children.

The results of the general physical examination were entirely negative, except for the pelvic examination. The uterus was markedly retroflexed; there were no palpable tumor masses.

Studies of the blood, the Wassermann and the Kahn reactions and urinalysis gave entirely negative results.

In the absence of other findings it was concluded that the relaxation of the pelvic floor, together with the marked retroversion of the uterus, had caused protrusion of the cervix into the bladder; this in turn had caused urethral obstruction.

On Feb. 21, 1931, laparotomy revealed marked retroversion of the uterus and a lemon-sized mass in the anterior wall of the large intestine, at the rectosigmoid junction. Ventral fixation of the uterus was done, together with ligation of the fallopian tubes and removal of the mass in the rectosigmoid wall. This mass was found to contain four cherry-sized diverticula, each containing masses of inspissated feces. There was no evidence of malignancy. A small fecal fistula developed on Feb. 26, 1931; it gradually closed during the next two weeks. The operation was followed by complete relief of symptoms. The patient has experienced excellent health since the time of operation.

Case 4.—Miss J. F., a school teacher, aged 55, was first seen on March 28, 1931. Two years previously, 100 Gm. of radium, with a brass screen, had been placed in the uterine cavity for twenty-four hours, in order to control uterine hemorrhage due to the presence of a submucous fibromyoma. In January, 1930, a

complete diagnostic survey was carried out. The patient's complaints at that time were marked constipation and pain in the lower part of the back, with a constant dull headache and insomnia. The pain in the back frequently radiated around to the right side of the abdomen.

The results of complete physical and laboratory examinations at this time were essentially negative, except that a barium enema revealed several small diverticula in the sigmoid colon.

On March 28, 1931, the patient experienced a sudden, sharp, generalized abdominal pain that gradually localized itself in the right lower abdominal quadrant. The onset of the pain was followed by nausea and vomiting. The temperature was 101.4 F., with a pulse rate of 105. The results of a general physical examination were negative, except for the presence of tenderness and rigidity of the muscles over the lower right abdominal quadrant. There were no palpable tumor masses. The blood picture showed a white blood cell count of 14,500 with 94 per cent polymorphonuclear neutrophils. Urinalysis gave negative results.

A diagnosis of acute appendicitis was made, and the abdomen was opened through a split-muscle incision. The cecum was found to lie in the pelvis, and the appendix was found to be normal. At the upper end of the incision a firm, spherical mass, 8 cm. in diameter, was palpated. When this mass was withdrawn from the abdomen, a small amount of pus escaped. The mass was found to be the hepatic flexure of the colon, with multiple small diverticula protruding from it. The mass was left outside of the abdomen, and dressings of hot boric acid were applied. Several of the diverticula ruptured and drained fecal material for several days. The inflammatory process subsided in about two weeks, after which the colon was replaced beneath the abdominal muscles, external to the peritoneum. The wound healed rapidly, and the patient has experienced good health since the operation.

SUMMARY AND CONCLUSIONS

1. Diverticulitis of the colon is a much more common disease than is generally appreciated.

2. Diverticulitis of the mobile sigmoid colon may produce symptoms of pelvic disease. In three of the four cases that form the basis of this thesis, the symptoms led to confusion with gynecologic conditions.

3. In one instance, reported herewith, the symptoms of acute diverticulitis closely simulated those of acute appendicitis.

4. Acute diverticulitis and the complications of chronic diverticulitis require surgical treatment. The withdrawal of the loop of bowel containing the infected diverticula through a split-muscle incision in the anterior abdominal wall, followed by the application of hot boric acid dressings until the inflammatory process has subsided, has yielded the best results in my experience. After the inflammation has subsided the loop of bowel may be released and placed beneath the abdominal muscles, external to the peritoneum. In the absence of intestinal obstruction or of evidence of carcinoma, it is rarely necessary to perform a Mikulicz resection.

FIFTIETH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY

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(Concluded from page 932)

MISCELLANEOUS

Echinococcus Disease of the Bone.—Coley [24] reported 2 cases of echinococcus disease of the bone. Both involved the bones of the pelvic girdle. In both cases the diagnosis was arrived at late in the disease and after the patients had been treated with high voltage roentgen therapy without benefit. In each case the diagnosis was made by biopsy. One patient is still alive, but the other died of complications following biopsy. Two forms of treatment are discussed. One consists in evacuation of the large cystic cavity and treatment with phenol or formaldehyde in the hope of producing fibrosis and subsequent obliteration of the cavity. The other, more ideal one consists in wide excision or amputation of the bone involved. The author stated that only 3 cases of osseus hydatid disease had been reported in this country up to 1930.

Roentgen Signs of Infantile Scurvy.—Kato [25] discussed the various so-called pathognomonic roentgen signs of infantile scurvy. The white

24. Coley, B. L.: J. Bone & Joint Surg. **14**:577, 1932.

25. Kato, K.: Radiology **18**:1096, 1932.

line in the metaphyseal area of the long bones seen in scurvy is also found in lead poisoning, phosphorus poisoning and other diseases. The contiguous area of diminished density of bone is likewise not pathognomonic. The sharp hairline outline of the epiphysis, while always present, is found in some other lesions. From a roentgen standpoint all of the so-called pathognomonic signs of scurvy must be correlated, and subperiosteal hemorrhage must be present if one is to make a positive diagnosis.

[ED. NOTE.—This article merits careful reading. The old conception of certain findings in certain diseases as classic must again be modified by reasoning and clinical observation.]

Diagnosis of Amyloid Disease.—In 1923, Bennhold discovered that an intravenous injection of aqueous solution of congo red could be used as a test for the presence of amyloid disease. Amyloid material absorbed the congo red, and the percentage decrease of the dye content of the serum over a period of one hour was estimated colorimetrically. In the normal person, from 10 to 20 per cent was found to have disappeared; in patients with amyloid disease, the diminution was between 30 and 100 per cent. Wallace [26] tried the test on 30 patients. It confirmed the diagnosis of amyloid disease in 15 of 17 patients clinically suspected of having the disease. Wallace argued that the test gave a quantitative result, and therefore was superior to Waldenstrom's liver puncture test, besides being less drastic.

Kienböch's Disease.—Mouat, Wilkie and Harding [27] studied 12 patients with Kienböch's disease. In 6 the semilunar bone was excised; of these, 4 resumed their former work, 1 did light work, and 1 could not be traced. In the other 6 the semilunar bone was not excised; of these, 1 resumed his former work, 1 did light work, and 4 remained unable to work. After removal of the semilunar bone, pain was relieved, and the outlook as regards recovery of function was excellent in the cases in which the prognosis was not impeded by a question of litigation. There was, however, some loss of range of movement, which, as a rule, diminished the wage-earning capacity.

Subchondral Focal Necrosis of the Third Metacarpus.—Dietrich [28] reported 7 cases of necrosis of the distal end of the third metacarpal bone which he believed were examples of aseptic necrosis. This would

26. Wallace, J. E.: Lancet **1**:391, 1932.

27. Mouat, T. B.; Wilkie, J., and Harding, H. E.: Brit. J. Surg. **19**:577, 1932.

28. Dietrich, H.: Arch. f. klin. Chir. **171**:555, 1932.

constitute a syndrome comparable to Köhler's disease of the second metatarsus. The clinical signs were mainly pain, local swelling of the affected joint and a sense of tightness on making a fist. Histologic examination of the bone of the first patient, a girl of 15 years, yielded a pathologic picture which fitted in well with that of aseptic necrosis. Some of the other cases were open to question as to the assuredness of the diagnosis. The last patient presented deformity of the metacarpus, broadening vacuolization and free body formation. Unfortunately no section of tissue was available in this instance.

Osteochondritis Deformans Contrasted with Osteochondritis Deformans Juvenilis.—In discussing hypertrophic arthritis and the changes in the joints associated with coxa plana (Legg's disease), Lang [29] concluded that although there are factors involved in the former, such as age, vascular changes and perhaps systemic disease, that are not present in the latter, trauma is an important causative factor in both, and that the disease originates after damage to the articular cartilage. This loss of elasticity removes the protection that the normal cartilage affords to the subchondral bone against functional and mechanical influences.

Disturbances of Consolidation After Orthopedic Osteotomies.—In 621 osteotomies performed during a ten year period, 8 cases of delayed union were encountered by Brandes.[30] Of these, 3 ended as true pseudarthrosis. In all but 1 case (pregnancy) local causes were present. Most of the disturbances occurred following osteotomies of the leg. No difficulty was encountered after osteoclasis.

TRAUMATIC INJURIES TO THE JOINTS

Traumatic Dislocation of the Hip.—Platt,[31] after remarking on the fact that traumatic dislocation of the hip is usually reduced without special difficulty and results in perfect function of the joint, recorded 4 cases with complications, as follows: (1) Fracture of the acetabular rim with extrusion of the loose bony fragment *per vaginam*: result, perfect. (2) Involvement of the sciatic nerve and myositis ossificans, necessitating excision of the head of the femur. Severe irritative signs developed as the sciatic nerve was recovering. The ultimate fate of the patient was unknown. (3) Chip fracture of the head of the femur and fracture of the acetabular margin, giving rise to two loose bodies. These bodies were removed, and the function of the hip joint was perfect, but only partial recovery occurred in the sciatic nerve, which was also damaged at the time of the original accident. (4) Chip fracture

29. Lang, F. J.: J. Bone & Joint Surg. **14**:563, 1932.
30. Brandes, M.: Arch. f. klin. Chir. **170**:408, 1932.
31. Platt, H.: Brit. J. Surg. **19**:601, 1932.

of a portion of the head of the femur giving rise to a loose body. This body was removed, and the result was perfect.

Injuries to the Tibial Collateral Ligament of the Knee Joint.—Regele [32] stated that injury to the tibial collateral ligament of the knee joint is the commonest injury in this region. The injury occurs through bending, which separates the upper and lower joint surfaces; through stretching, which can be of several types, and through side bending, which produces an increased valgus. In the milder cases there is a sharp pain immediately or pain only after a number of hours. The knee is usually held in slight flexion; there is some swelling on the inner side of the knee. In the more severe cases hemorrhage into the joint of the knee is the rule; pain is not as definitely localized as in the milder cases. Complete extension of the knee is restricted chiefly. Lateral stretching of the knee causes pain referred to the tibial collateral ligament. Sometimes there is an abnormal amount of lateral motion. These severer injuries are frequently associated with changes in the internal meniscus. The differentiation between an injury to the meniscus alone and an injury to the tibial collateral ligament is shown by more severe pain immediately in the former and slowly developing pain in the latter. The localization of the pain is helpful. Pain occurs on attempted adduction of the knee only when the ligament is affected. Hemorrhage into the joint is usually the result of an injury to the ligament. There is greater hindrance to extension when the meniscus is injured. Roentgenograms often show bony spicules torn off in injury to the tibial collateral ligament, while they show no changes in the meniscus. Severe tears may lead to an unstable knee and to arthritic changes. Bowing sometimes develops, more so in the heavy patient. Later limitation of motion may develop from faulty healing of the ligament. For treatment the author advises fixation of the knee in extension in a plaster cast. Hemorrhages should be aspirated immediately, and a pressure bandage applied before the cast is put on to avoid a recurrence of the hemorrhage. Fixation should be continued for from four to eight weeks, according to the severity of the injury, the constitution and the age. In young persons from one to two weeks is often sufficient. Walking in the cast is permitted immediately. Bad results occur from improper fixation, fixation in flexion or hyperextension, failure to remove the hemorrhage and early massage, which tends to increase the bleeding.

Tennis Elbow.—Carp [33] advocated a new form of treatment for epicondylitis (tennis elbow). The treatment consists in applying firm

32. Regele, H.: München. med. Wchnschr. **79**:1474, 1932.

33. Carp, L.: Tennis Elbow (Epicondylitis) Caused by Radiohumeral Bursitis: Anatomic, Clinical, Roentgenologic and Pathologic Aspects, With a Suggestion as to Treatment, Arch. Surg. **24**:905 (June) 1932.

pressure with the thumb to the region of the bursa over the radiohumeral joint. Such pressure is maintained for two or three minutes until it is felt that the bursa has been ruptured and its contents allowed to escape into the surrounding tissues. In some cases the relief from pain is striking. The anatomic, clinical, roentgenologic and pathologic aspects of the condition were reviewed, and the details of 8 cases were given. Four of the patients were treated by applying digital pressure on the bursa. All recovered almost immediately, and there were no recurrences. One patient was treated by operation, and the others were given physical therapy, and splinting was applied.

OPERATIONS ON THE BONE, JOINT AND TENDON

Preliminary Implantation of Bone in Pseudarthrosis.—Mayeda [34] applied the method proposed by Axhausen in 1928 to infected pseudarthroses. It is of value when one wishes to save the time which ordinarily is permitted to elapse between the healing of an infected pseudarthrosis and the date when a bone graft operation can be performed. The infected pseudarthrosis is drained and permitted to heal. Soon after healing, possibly even while a granulating area persists, a tibial or preferably a costal segment covered with periosteum is implanted in the healthy soft tissues immediately adjacent to the site of future bone grafting. This graft is permitted to develop a blood supply. At the next step, the graft is exposed over one aspect and brought in contact with the freshened pseudarthrosis and fastened in place with wire as a massive onlay graft. If infection develops, it obviously will not destroy such a vascularized onlay graft.

Dupuytren's Contracture.—Davis [35] reviewed the results of the treatment of Dupuytren's contraction in 42 cases, as follows: Excision of fascia was performed in 31 cases, with 12 perfect results, 10 partial returns of the contracture, 3 complete returns of the deformity and 6 anatomically perfect results that were functionally useless on account of painful scar, digital anesthesia or both, causing total incapacity. Multiple subcutaneous division was employed in 8 cases, with 6 perfect results, 1 partial return of the contracture and 1 complete return of the contracture. Injection of fibrolysin was tried in 3 cases, with no perfect results, 2 partial returns of the contracture and 1 complete return of the contracture. Davis concluded that as the guarantee of cure remained far from certain even following major operative procedures, the minor operation of multiple subcutaneous fascial division should be adopted, and repeated if necessary.

34. Mayeda, T.: Deutsche Ztschr. f. Chir. **236**:159, 1932.
35. Davis, A. A.: Brit. J. Surg. **19**:539, 1932.

[Ed. Note.—Some of the editors take exception to the author's conclusions. Excellent results can be obtained by thorough excision of the contracted tissue, but in some instances this must be followed by plastic operations to close defects in the skin. Successful results by this method have been reported by many authors.]

Approach to the Anterior Portion of the Elbow Joint.—Gerlach [36] reported the end-results of 28 operations on the anterior portion of the elbow joint, using the operation devised by Läwen.[37] The operation was performed through an anterior midline incision over the elbow joint; the tendons of the biceps and the brachialis muscle were split. This operation was found particularly useful in the removal of foreign bodies from the anterior compartment of the elbow joint, and in the open reduction of fractures with anterior displacement of fragments. The author stated that there was no danger of injury to the nerve in this operation, and that the only blood vessel encountered was the medial cubital vein, which was ligated in each instance. For foreign bodies much displaced ulnad, a small transverse incision in the longitudinally split capsule gave adequate exposure. In the latter operations the joint capsule was not sutured; the author believed that the drainage which this permitted was advantageous. Primary healing was observed in all cases. Physical therapy was begun in from eight to ten days. In 19 cases in which the end-results were obtainable, there were 10 excellent results and 1 ankylosis.

Recurrent Inferior Radio-Ulnar Dislocation.—Eliason [38] described a new operation for recurrent inferior radio-ulnar dislocation. A strip of fascia was passed around the lower end of the ulna, and then both ends of the fascia were carried through a drill hole in the radius running from the posterior and ulnar aspect forward and outward to penetrate the outer radial cortex about 1¼ inches (3.14 cm.) above the radial styloid. It was necessary to perform a secondary operation on the same wrist six months later because of a severe strain produced by sharply supinating the wrist. At the second operation the anterior and posterior radio-ulnar ligaments were plicated to take up the relaxation of this structure. In addition, a portion of the ulnar attachment of the pronator quadratus muscle was shifted distally and sutured over the joint as an additional splint. The author recommended that both procedures be carried out at one sitting.

[Ed. Note.—This seems to be a rather elaborate procedure. One should consider whether subperiosteal resection of the lower end of the ulna as advocated by Darrach would not give a better functional result.]

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36. Gerlach, K.: Beitr. z. klin. Chir. **155**:589, 1932.
37. Läwen, A.: Arch. f. klin. Chir. **162**:521, 1930.
38. Eliason, E. L.: Ann. Surg. **96**:27, 1932.

Operative Fusion of Unstable or Flail Knee in Poliomyelitis.—Cleveland [39] presented a study of 90 patients with infantile paralysis on whom operative fusion for flail legs had been performed. The youngest patient was 9 years of age; the oldest, 33 years; the average age was from 15 to 16 years. In practically all cases the reason for fusing the joint was to obviate the use of a brace. Practically no increase in shortening was obtained after operation, it being on an average 2 inches (5 cm.) as compared to 1¾ inches (4.37 cm.) before operation. In all but 2 patients fusion was obtained at the first operation. Flexion and knock-knee deformity occurred in 15 patients following the operation and were due to hamstring contracture and ununited epiphyses. These complications required secondary corrective operations. In 85 per cent of the patients good cosmetic and functional results were obtained.

FRACTURES

Fractures in and Near the Ankle Joint.—Miller,[40] in discussing fractures in and about the ankle joint, stated that reduction is usually quite easy if undertaken in the first few hours after the fracture occurs. The most satisfactory anesthesia is from 10 to 15 cc. of a 2 per cent solution of procaine hydrochloride injected into the hematoma about the fracture. It is most important to secure the normal relationship of the articulating surfaces of the ankle joint. Immobilization is maintained by a plaster cast which is bivalved for physical therapy in one week. Weight bearing is not permitted until firm union of the fracture has occurred, which is usually in eight weeks.

Fractures and Epiphyseal Separation at the Ankle.—Bishop [41] reviewed the classification of fractures of the ankle proposed by Ashhurst and Bromer, i. e., according to the mechanism of production, and classified 300 cases in this way: (*A*) fractures caused by external rotation, 54.4 per cent; (*B*) fractures caused by abduction, 26.3 per cent; (*C*) fractures caused by adduction, 14 per cent; (*D*) fractures caused by compression, 4 per cent, and (*E*) fractures caused by direct violence, 1.3 per cent. His percentages in the various groups were almost identical with those given by Ashhurst. He then reviewed 32 fractures associated with separation of the lower tibial epiphysis and classified them in the same way: (*A*) 18.7 per cent, (*B*) 12.5 per cent, (*C*) 53.1 per cent, (*D*) 12.5 per cent and (*E*) 3.2 per cent. There were incomplete data on the end-results, indicating that deformities of growth from injury to the epiphysis were rare.

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39. Cleveland, M.: J. Bone & Joint Surg. **14**:525, 1932.
40. Miller, O.: Kentucky M. J. **30**:407, 1932.
41. Bishop, P. A.: Am. J. Roentgenol. **28**:49, 1932.

Fractures of the Shafts of the Tibia and Fibula.—Jones [42] described a new type of apparatus for reducing fractures of the tibia and fibula. It consisted essentially in a thigh support which could be fastened to any table over which the leg hung at a right angle. A pin was passed through the os calcis; a stirrup was fastened to the pin, and the stirrup was then fastened to a traction bar, counteraction being gained against the thigh support. As traction was maintained, alinement was perfected by manipulation of the fragments. A nonpadded cast was then applied from the toe to the tibial tubercle. As the plaster dried, the knee was straightened to an angle of 20 degrees of flexion, and the plaster was carried to the groin.

Fractures of the Shafts of the Long Bones.—Schaanning [43] reviewed the treatment of 260 fractures of the shaft of the long bones between 1919 and 1928 at the Rikshospital, Oslo, Norway. Of these fractures, there were 113 in the lower leg, 89 in the thigh, 30 in the upper arm and 28 in the lower arm; 46 were compound fractures. Eighty patients were treated by operation, and in the remainder the fractures were reduced by manipulation. Operations were performed most frequently on the forearm, in 53.5 per cent of this series. Operation was usually performed in the first or second week after the injury. The indications for operative intervention were: (1) interposition of muscle or nerve, (2) possibility of injury to the blood vessels, (3) irreducible dislocation, (4) multiple fractures in the shaft of a bone, (5) danger of fusion of two adjacent bones and (6) failure to unite in a reasonable length of time. Operative reduction was undertaken on all fractures of the patella and olecranon. The period of convalescence averaged seven months in the nonoperative cases and ten and three-fourths months for the patients who were operated on. The methods of operative reduction were as follows: plating with the Lane or Lambotte plate, replacement of the fragments, encircling by the Parham band, wire suture, use of an ivory peg in 1 case and bone grafting in 1 case. The author believes that operative intervention is an admirable supplement when nonoperative manipulation fails. The best results were obtained when operative intervention was undertaken early. Compound fractures were an exception to this, since early operation often caused widespread infection. Operations were unusual on children under 10. In elderly patients the general condition of the patient was of more importance than the age. In operative fixation the author found the Lane plate the most satisfactory measure. He did not believe that the foreign body hindered bony union. There were 2 deaths, 1 probably due to fat embolism. Infection occurred after operation in 8 patients; 7 of these had com-

42. Jones, Robert: J. Bone & Joint Surg. **14**:59, 1932.
43. Schaanning, C. K.: Acta chir. Scandinav. **70**:1, 1932.

pound fractures. In a few cases (number not given) the Lane plate had to be removed because of persistent tenderness.

Fractures of the Neck of the Femur.—Gill [44] presented an excellent summary of the various methods of treatment of fractures of the femoral neck and deplored the fact that it is impossible to compare statistics because in some clinics cases are selected and in others they are not. He dealt with the more common methods of closed and open reduction and evaluated each fairly. He also discussed the treatment in cases of nonunion. He held that in certain cases operative fusion of the hip gives the best results.

Autogenous Bone-Graft Peg in Fresh Fractures of the Femoral Neck.—Albee [45] described his technic for the open reduction and pegging with an autogenous bone graft of ununited and fresh fractures of the femoral neck. The author stated that as nonoperative methods had shown such poor results and as the operative management was not tedious or hazardous, all fresh fractures of the femoral neck should be treated by autogenous bone pegging. This procedure was stated to give excellent results.

[ED. NOTE.—We believe that the treatment of fractures of the neck of the femur represents a complex problem. Operative treatment is permissible in the hands of good surgeons, but it has not yet been proved that better results are obtained than by the Whitman method of treatment.]

Treatment of Compression Fractures of the Vertebrae by Roger's Frame.—Mitchell [46] advocated the use of Roger's frame in treating compression fractures of the spine. Patients were kept recumbent in hyperextension after reduction; then a brace was worn for from three to six months longer. Reduction was sometimes accomplished one month after injury. Laminectomy was indicated only in the presence of neurologic findings and a positive Queckenstedt test.

Fractures of the Cervical Vertebrae.—Ten cases of fracture of the cervical vertebrae were reported in detail by Laesecke.[47] Conservative treatment was used even when injuries of the cord were present. Not infrequently complete healing took place. He warned against too early resumption of weight bearing on the injured spine.

Fractures of the Pelvis.—Leadbetter [48] discussed the treatment of fractures of the pelvis, basing his conclusions on a study of 100 cases. He stated that no one type of treatment was applicable to any type of

44. Gill, A. B.: Ann. Surg. **96**:1, 1932.
45. Albee, F. H.: California & West. Med. **37**:1, 1932.
46. Mitchell, J. I.: South. M. J. **25**:473, 1932.
47. Laesecke, M.: Deutsche Ztschr. f. Chir. **236**:329, 1932.
48. Leadbetter, G. W.: South. M. J. **25**:742, 1932.

pelvic fracture; each represented a problem in itself. The aim in each case was to bring about as perfect a restoration of the pelvic ring as possible. Only in this way could the late sequelae resulting chiefly from faulty body mechanics be avoided. The author found that a fracture of one or both pubic rami was the commonest type of fracture. Fractures of the upper third of the iliac crest were next most common. Separation of the sacro-iliac joints with displacement of the ilium was seen in 10 per cent of the cases. It was noticed that when multiple fractures occurred they all tended to be in the straight line. The author found congenital anomalies of the lower part of the spine or the pelvis in 67 per cent of the cases, but he was unable to state whether they were a predisposing factor in the causation of the fractures. The author used suspension-traction most commonly. Comfort and easy reduction of the fractures were observed in almost all of the patients so treated. In cases in which reduction was not accomplished in forty-eight hours, skeletal traction was used. Traction was continued for six weeks, and the patient was kept on a plaster board for two weeks more. Weight bearing was permitted in ten weeks.

RESEARCH

Effect of Parathyroidectomy and the Administration of Parathormone on Bone Repair.—Dragstedt and Kearns [49] excised a small segment of bone from the femur in 20 dogs and noted that the rate of repair was quite uniform unless distemper or diarrhea developed. On an additional 22 dogs thyroparathyroidectomy was performed after the femoral defect was produced. The development of tetany was controlled by the administration of calcium. Thirteen dogs did not survive the necessary sixty days of observation. In the 14 dogs which remained in comparatively good health and had received from ½ to 1½ units of parathormone per kilogram per day, the rate of healing of the femur was essentially the same as in the control series.

Intrinsic Circulation of the Vertebral Body.—Wagoner and Pendergrass [50] investigated the circulation of the blood within the vertebral body in the lumbar spine. The chief blood supply was from the lumbar arteries, the branches of the abdominal aorta. On each side of the vertebral body there was an anterolateral and a smaller lateral artery. A dorsal branch sent many small arteries directly into the bone. This dorsal artery divided at the intervertebral foramen into a spinal branch and a muscular branch. Each vertebral body was drained by four venous trunks, one vein leaving from each side of the body and two

49. Dragstedt, C. A., and Kearns, J. E., Jr.: Experimental Study of Bone Repair: Effect of Thyroparathyroidectomy and of the Administration of Parathormone, Arch. Surg. **24**:893 (June) 1932.

50. Wagoner, G., and Pendergrass, E. P.: Am. J. Roentgenol. **27**:818, 1932.

from the foramen in the center of the posterior vertebral wall. The lateral veins emptied into the lumbar veins, and the posterior veins emptied into the anterior longitudinal meningorachidian veins. The vertebrae were found to contain much blood, which had a sluggish rate of flow. There was a large venous reservoir in the midportion of the vertebral body. Here the normal venous wall was replaced by a single layer of flattened endothelial cells. The large venous channels probably played a rôle in the formation and destruction of blood; they removed and destroyed bacteria and were a factor in the localization and extension of disease processes in the vertebrae.

Roentgen Spectrographic Investigation of the Crystalline Structure of Bone.—Henschen, Straumann and Bucher [51] found that roentgen rays might be used like visible light to study the structure of material by the spectra produced. Such studies had been performed by the authors, bone being used. Both normal and pathologic bones were subjected to study. The significant findings were the verification of the crystalline structure of the inorganic salts of the bone. The organic portions of bone also show evidence of crystalline structure. The two portions of bone, the salts and the fibrils, exhibited absorption in their relationship. Also, since the character of bone changed materially with advancing years, the authors suggested that the age of bone might be approximated from spectrographic plates.

51. Henschen, C.; Straumann, R., and Bucher, R.: Deutsche Ztschr. f. Chir. **236**:485, 1932.

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